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RESULTS OF THE USE OF THE DRINKER RESPIRATOR IN THIRTY CASES OF RESPIRATORY FAILURE IN POLIOMYELITIS*

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RESPIRATORY failure in poliomyelitis may be ushered in in a variety of ways depending on the locality and extent of the pathology. It is well to bear in mind that the disease is a generalized infection which has gained access to the central nervous system, and that the effects of the general infection in the early stage may be either negligible, or in some instances debilitating. Furthermore, evidence of motor paralysis in fibres of one or more cranial or spinal nerves not connected with respiration often precedes evidence of respiratory failure. Consequently the respiratory symptoms are superimposed on a variety of other disturbances remotely or closely associated.

At this point it is well to discuss briefly the possible sources of injury in poliomyelitis which may bring about respiratory failure. The primary effect of the virus, once it gains access to the central nervous system, is to produce a proliferation of the mononuclear cells lining the meningeal vessels. These vessels feed and drain both the medulla and cord. In connection with respiratory failure, we must bear in mind that the nuclei of the glosso-pharyngeal and vagus nerves lie in the medulla, and the motor nuclei of the nerves supplying the essential respiratory muscles lie in the anterior horns of the cervical and dorsal portions of the cord. The respiratory center itself lies in the medulla under the floor of the fourth ventricle.

The injury to these nerve nuclei in the medulla and cord may be brought about in several ways. The proliferation of the lining cells may occlude the blood supply and starve the nerve cell; this proliferation may also obstruct the outflow in the veins; or the virus may directly attack the nucleus itself, which action would be augmented by venous obstruction. Such a disturbance of the meningeal vessels constitutes a type of inflammation which tends to increase the amount of spinal fluid, and tends to throw off into this fluid a preponderance of mononuclear cells. Furthermore, in those areas affected within the medulla and cord we get a variable degree of actual edema depending on the amount of inflammation. The possibility of hemorrhage in these areas, with its serious local consequences, need only be mentioned. Depending on the type and degree of injury we may have temporary or permanent, and partial or complete paralysis of a muscle or group of muscles.

One of the very early manifestations of cranial nerve involvement is palatal paralysis with the characteristic gutteral speech. If severe enough, the paralyzed palate allows fluids to return through the nose on swallowing. Paralysis of the palate alone does not necessarily impair respiration except by causing choking and coughing when the fluid runs back from the nose after the act of swallowing is completed. But we must remember that swallowing is a complicated process requiring coördination and rapidity of action on the part of several muscles. When other muscles besides those of the soft palate become paralyzed, the danger to the respiratory apparatus becomes much greater through improper closing off of the larynx, which allows the inspiration of fluids, food particles, or mucus into the trachea and bronchi.

The phrenic nuclei lie in the cervical section of the cord. Thus the diaphragm is liable to be affected when this segment of the cord is involved. The essential respiratory muscles of the neck and thorax get their motor nerve supply from the spinal nerves, the nuclei of which—as we have already said—lie in the cervical and dorsal portions of the cord.

From the above brief outline one has at least a pathological and anatomical basis for classifying the various types of respiratory failure in poliomyelitis. In actual practice this is not always easy because of the combinations sometimes presented in a single case. As examples of this difficulty we present three cases of serious respiratory embarrassment in which the actual type of injury remains uncertain because of its temporary character.

A boy* of fifteen months, admitted to our wards in 1927 before we had respirators, showed paralysis of all four extremities and no apparent chest expansion with inspiration. The abdominal reflexes were absent, and respiration was being carried on by the diaphragm. There was difficulty in swallowing with slight regurgitation through the nose, and a very weak cry. No other symptoms of cranial nerve involvement were present. Perspiration was profuse. There was no vomiting after admission. Constipation was relieved by enemas. On the fifth day he

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*Hospital Case No. 18671, Sept. 13, 1927. Dr. W. B. Christie, senior interne at the time. No serum administered.

began coughing, became cyanotic, and almost stopped breathing. Lumbar puncture was immediately done, and the spinal fluid was allowed to squirt off under great pressure. The needle was then removed and artificial respiration begun, as the baby had stopped breathing. With continued artificial respiration, diaphragmatic breathing was gradually resumed. Periodic impairment of respiration recurred every six or eight hours, and this continued with lengthening intervals for ten days. Lumbar puncture and drainage were resorted to whenever the respiratory failure seemed imminent, and increased pressure was always present. On several occasions continued artificial respiration had to be resorted to after the puncture. There was no struggling during the drainage. A suction tube for the pharynx was never needed, as there was no mucus accumulation. During the ten-day period swallowing and crying became normal. Eventually the thoracic muscles regained their power, and the baby was discharged with badly paralyzed extremities on the seventy-first day, to die six months later from broncho-pneumonia during a severe chickenpox.*

We were under the impression that drainage was the essential life-saving measure, but, owing to this prejudice, we never applied artificial respiration beforehand, so that we can only speculate on the relative value of these two measures in this case. It is impossible to say whether there was edema in the medulla or in the cord, but there was an excessive amount of spinal fluid formation. There was evidence of cranial nerve involvement, but this improved during the period. Consequently, we are not justified in assuming that an edema of the respiratory center existed. In the light of subsequent experience we feel that this case would have done better in a respirator. This case, however, does show what could be done without one.

Another case is that of a girl of twelve† admitted to the hospital on August 22, 1931. She had been taken ill on August 18 with nausea and vomiting, which subsided. On the 22nd she was found to have headache, stiffness of the neck and back, a fever of 101° F., and difficulty in swallowing. On arrival at the hospital she showed T. 102°, P. 80, R. 30, and in addition to the above, palatal paralysis, partial pharyngeal paralysis, with a thick mucus collection in the throat which she expectorated at times, but with inability to cough. Suction was applied to the throat without much relief. There was good chest expansion, and normal diaphragm excursion as evidenced by the abdominal bulge on normal inspiration. Knee jerks absent, biceps and triceps jerks diminished, abdominals normal. The rest of the physical examination showed no abnormalities worthy of mention here. The spinal fluid was clear and under a pressure of 17 mm. Hg., cell count 50, sugar present, and globulin increased. She was given glucose by rectum every three hours. By evening the temperature was 99°, pulse 86, and respiration 38, and she was irrational at times during the night. The next day respiration became irregular and jerky, but this subsided after lumbar drainage. On August 24 she began to be able to swallow with care, and by midnight had taken 30 ounces. Rectal glucose was continued and well retained. Restlessness and jerky respirations returned at 10 A. M. on August 25, and after lumbar drainage of 25 cc. (under an initial pressure of 22 mm. Hg.) the patient became quiet and the breathing regular at 36-30 (P. 86). During the evening the respiration gradually slowed, becoming markedly jerky. At 10:50 P. M. the respirations dropped to 10. The interne was called, and by the time he arrived she had stopped breathing and was cyanotic. All the four respirators on the floor were occupied. With artificial respiration she began to breathe again. Lumbar drainage was then done. Such marked relief followed that she was not put in a respirator. At 5 A. M. (August 26) the temperature was 98°, pulse 78, and respiration 20. At 8 A. M., she was placed in the respirator for five hours, when she was removed, given a sponge bath and an enema. She was put back for four-hour periods during the next three days. At 8 P. M. on August 27 she coughed for the first time. That night there was a heavy sweat. She now began taking liquids in large amounts. On August 28 she took soft solids with relish, but hiccoughed moderately while taking her food. Respirations remained otherwise normal. There were no râles. She was discharged on the fifteenth day (September 5) with no signs of paralysis; completely recovered.

This was an unusual case in our experience because at no time was there any evidence of spinal nerve involvement, and yet artificial respiration restored breathing. Of course, from our observations it is impossible to do more than speculate as to why breathing stopped. This case is included among those that might have survived without the respirator, yet this machine played an important part in aerating the lungs and avoiding anoxemia. It will be noted that the lumbar drainage was done after artificial respiration, and that improvement followed. Whether this improvement was in any way related to the drainage is questionable. However, we do feel that on negative lines alone these two cases afford a refutation to Macnamara's* warning against lumbar drainage without serum replacement.

A third case† gives an example of the efficacy of the prompt use of the respirator. A girl of seven was admitted at 7 P.M. on September 25, 1930, with a diagnosis of preparalytic poliomyelitis. (B. P. 135/90.) Lumbar puncture was done immediately, 28 cc. of spinal fluid was withdrawn (pressure 9 mm. Hg., cell count 190), and replaced with 20 cc. convalescent serum. An additional 60 cc. of this same serum was given intravenously. Within one hour of admission she began to show some difficulty in swallowing, which continued throughout the night, yet by taking fluids slowly she got in 28 ounces between 7 P. M. and 6 A. M., after which she drank freely and easily. During the night the interne noticed a diminished expansion of the left side of the chest when the patient was asked to take long breaths. On September 26 there was profuse sweating. She complained of dizziness throughout the day, and became less responsive. Both arms and the left leg became paralyzed during the afternoon. Catheterization was necessary. (B. P. 140/85.) Spinal drainage of 28 cc.; cell count 2500. At 6:30 P. M. the

*The fact that the baby died six months later from a broncho-pneumonia with chickenpox suggests that the broncho-pneumonia might have resulted from remaining respiratory weakness from the poliomyelitis.

†Hospital Case No. 23400. Dr. R. W. Rutherford, senior interne. No serum administered.

*Macnamara, J.: The treatment of acute poliomyelitis by means of human immunizing serum. Med. Jour. Australia, 16:2:24, 838 (Dec. 14), 1929.

†Hospital Case No. 21924.

costal† muscles became completely paralyzed, and she suddenly began to gasp for air. She was immediately placed in a respirator where she slept all night. Spinal drainage was done on the 27th. (B. P. 125/80.) The next day B. P. was 110/75. No further lumbar punctures were done. She remained in the respirator most of the time for two weeks, at the end of which time she could be taken out for as long as twelve hours. She was discharged on the 130th day with good chest expansion, and only partial paralysis of the left arm, which continues to improve under orthopedic care.

There can be no doubt as to the value of the respirator in this case. Furthermore, once the thoracic muscles became incompetent, the diaphragm tired out so quickly that she was suddenly plunged into a serious condition of air hunger. It is very doubtful whether after the onset of respiratory failure she would have lasted long enough to reach a hospital. The failure in this case was of the spinal type and consisted of a complete bilateral costal paralysis with sudden weakening on the part of the diaphragm. However, she was able to get along for short periods without the respirator because of regained control of the diaphragm, and she eventually made a good recovery, not only of the rib-raising muscles, but of the extremities as well. The question arises as to whether she would have done better if placed in the respirator during the first day after the irregularity in chest expansion was noted. This seems probable, but we were guided by the absence of any embarrassment to normal breathing.

Those unfamiliar with respiratory failure in poliomyelitis are apt to overestimate the value of the respirator in this disease because they fail to differentiate the types of respiratory failure. The pure spinal type has a very good chance of being saved by the machine if used promptly. On the other hand, in the pure bulbar type with glosso-pharyngeal and vagus involvement, the respirator often offers little help. Where coördination with the machine is impossible or where hiccough is present, the patient is often worse off in the respirator. Four out of five of the pure bulbar cases died. Thus in our series we find a high mortality in the pure bulbar type, but an even higher mortality in the ascending mixed type. The term is used here in the usual sense of the so-called "ascending type," which owing to the respirator lived until the cranial nerves were involved, while our use of the term "descending type" denotes that paralysis was first manifested in the cranial nerves and later in the spinal nerves.

During the years 1930 and 1931, there were admitted to the Haynes Memorial Hospital 170 cases of acute poliomyelitis, of which 30 were treated for respiratory failure in Drinker respirators. Thirteen of these 30 were admitted with respiratory failure, six being *in extremis* on admission. Of these thirteen, ten died in spite of the respirator. Seventeen cases developed respiratory failure subsequent to admission, and seven of these died. From this it appears that when respiratory failure comes on where a respirator is at hand the chances of recovery are much better.

We had hoped to draw some conclusions regarding the relative importance of diaphragm and costal paralysis, but in this series there were only four cases in which there was evidence of paralysis of the diaphragm, and in all four of these it was in combination with paralysis of the chest muscles. Whether the diaphragm gave out as a result of fatigue in trying to compensate for the paralyzed chest muscles, or whether the diaphragm succumbed to the same paralysis as the chest muscles, is difficult to determine. We know that the diaphragm may be paralyzed unilaterally or bilaterally, as evidenced by the x-ray* and fluoroscope, and in cases treated previous to 1930 we have seen paralysis confined to the diaphragm. When respiratory failure takes place with diaphragm paralysis we are usually not confronted solely with the loss of the diaphragm but also with a weakening of the group of muscles which coördinate with it. Consequently we cannot draw conclusions from these cases because of the difficulties encountered in attempting to interpret our observations on this point.

In Table I it is seen that all nine of the pure spinal cases treated with the respirator survived, one bulbar case out of five, three mixed cases of the descending variety, and none of the ascending variety. Since respiratory failure is the usual mode of death in acute poliomyelitis, and since during these two years we lost 21 cases from this disease, all with respiratory failure—four having died early in 1930 before we had respirators—it is safe to say that our hospital mortality has been reduced by more than one-third through the use of the respirator.

There is another factor pertaining to the evaluation of the respirator which cannot be shown in figures. If we had kept a record of the number of cases of respiratory failure for which our ambulances started but arrived at the house after the patient's death, or the number of cases that we were told were being rushed to the hospital but died before they got started, then we should have a better idea of what the prompt use of the respirator means in respiratory failure. To this perfectly definite value as a life-saving measure must be added another virtue which also cannot be shown by figures. For even in fatal cases, where coördination was possible, the extreme distress usually seen was relieved.

Included among the total thirteen recoveries

†We have purposely avoided referring to the intercostal muscles because so many muscles of the thorax are involved in raising the ribs, especially when any impairment leads to forced inspiratory efforts.

*Favill, J. and Fentress, T. L.: A case of respiratory paralysis in poliomyelitis successfully treated in a respirator. J. A. M. A., 97, 20, 1464, (Nov. 14), 1931.

are three cases which, in all fairness and judging from past experiences without respirators, might possibly have recovered without the aid of the machine. In our opinion, the other ten cases would have died without the respirator. There were a number of bulbar cases with complete loss of deglutition and embarrassment to respiration only because of the collection of mucus. These cases we found did better outside the respirator with occasional pharyngeal suction, and rectal and intravenous glucose, until nasal feeding was safe. This group was composed of older children and adults. Had these cases been included among our respirator series it would have given a false impression of the actual value of the machine.

TABLE I

RESPIRATORY PARALYSIS

	On Admission			After Admission			Total Cases		
	No. of cases	Recoveries	Deaths	No. of cases	Recoveries	Deaths	No. of cases	Recoveries	Deaths
Bulbar	3	0	3	2	1	1	5	1	4
Spinal	2	2	0	7	7	0	9	9	0
Mixed (bulbo-spinal)									
Descending	5	1	4	4	2	2	9	3	6
Ascending	3	0	3	4	0	4	7	0	7
	13	3	10	17	10	7	30	13	17

ADDITIONAL DATA

Might have recovered without respirator	3
In our opinion saved by respirator	10
Admitted *in extremis*	6
Died within 24 hours of being placed in respirator	12
Poor coördination. Hiccough and jerky respirations	11
"Dromedary" types	3

The condition on discharge of the thirteen who recovered offers an equal amount of satisfaction and discouragement. Two cases were discharged with no evidence of paralysis whatever. One of these might have lived without the respirator, but it is fair to assume that the paralysis might have become permanent with continued anoxemia. Eleven cases had paralysis of extremities on discharge, slight in two, moderate in one, and severe in eight. Indeed, it must not be understood that survival of these cases carries with it the promise of complete recovery. Two of the thirteen cases have a residual complete paralysis of both arms and both legs. On the other hand, four have shown marked improvement under the care of the orthopedic department.

Among these eight severely paralyzed cases we have included a boy* who arrived at the hospital with all four extremities completely paralyzed (a "dromedary" type). On the third day he developed respiratory failure (spinal, costal type), and was definitely saved by the respirator. On the tenth day he could stay out for half an hour. Eventually he got along without the respirator, but was placed in it daily for a few hours for six weeks to rest his respiratory muscles. On his 69th day he was ready for discharge to the orthopedic department with a fairly good chest expansion when the night nurse came down with a sudden coryza while on duty, and before she was relieved the patient contracted the infection, and developed broncho-pneumonia from which he died in two days. This case had recovered as far as the acute respiratory failure was concerned, and had been living without the respirator for a month when he got the cold. Consequently it is only fair to include him as a recovery in order to give the respirator due credit. Others have included these late deaths from broncho-pneumonia among their deaths. In a way this is right because the poliomyelitic paralysis predisposed them to the broncho-pneumonia. In this case, the patient would have been discharged two weeks before if arrangements could have been made. However, the secondary cause of his death was the poliomyelitis with respiratory paralysis. This is a striking example of the extreme susceptibility of these cases to broncho-pneumonia.

The value of convalescent serum in the preparalytic stage seems to us to be suggested in this small series. Seven cases admitted as preparalytics and given serum on admission subsequently developed respiratory failure, and only one died, a mortality of 14.2 per cent. The other twenty-three cases showed paralyses of one sort or another on admission, that is, they were already in the paralytic stage of the disease. Only three of these received serum, and these three died when respiratory failure followed. Twenty other cases admitted in the paralytic stage received no serum, and thirteen of these or 65 per cent died of respiratory failure. Realizing that the use of percentage figures is

*Hospital Case No. 23038, 1931.

precarious in such a small series, we nevertheless feel that the difference between 65 per cent and 14.2 per cent suggests that convalescent serum has a protective value when given in the preparalytic stage. One would suppose that if serum in the preparalytic stage can lessen the degree of subsequent respiratory failure, then serum ought to be able to work in this protective manner in the paralytic stage. Our three deaths do not support this view, but one can not draw conclusions from three cases. Unfortunately, the supply of convalescent serum does not permit of its general use in the early paralytic stage. The serum of the Harvard Infantile Paralysis Commission supplied to the Haynes Memorial has been reserved for the preparalytic cases where one can hope for more benefit from its administration, and better determine its efficacy.

TABLE II

CONVALESCENT SERUM TREATMENT BEFORE ONSET OF RESPIRATORY FAILURE

	No.	Died	Mortality percent
Serum Received			
In preparalytic stage	7	1	14.2
After onset in paralytic stage	3	3	100.
	10	4	40.
No Serum Received (Admitted in paralytic stage)	20	13	65

SUMMARY

We have discussed four cases of respiratory failure, and reported the results of thirty cases treated in the Drinker respirator at the Haynes Memorial Hospital. The emergency character of respiratory failure has been depicted. The results have shown that the respirator proved particularly effective as a life-saving measure in the spinal type, but to a much less extent in the mixed bulbo-spinal type and the bulbar type. Patients showing lack of coördination with the machine, jerky respirations, and severe hiccough were rarely benefited. When coördination was possible the respirator afforded great relief and comfort even in fatal cases.

THE CHIEF

Remarks to the First Graduating Class of the Medical Department of Duke University on the Occasion of the Planting of a Sprig of Ivy from 13, Norham Gardens

BY WILLIAM S. THAYER, M.D.*

IT is always a joy to be asked to speak about the "Chief" and it is an especial pleasure to be asked to take part in such an occasion as this, in such a spot as this, and at such a time. When I first came to know Osler it was under conditions like those which exist here today. The Johns Hopkins Hospital was then but little over a year old. Its traditions lay before it. Osler had come to Baltimore at its opening to organize the new medical department. The school of medicine had not yet been opened.

The son of the rector of a country parish in upper Canada, of good Cornish stock, Osler was destined at first for the clergy. But early in life he fell under the influence of two teachers, both clergymen, both fine, strong characters, both devoted to the natural sciences. Osler followed in their footsteps which led him also to the natural sciences, and 1870 found him in the McGill School of Medicine. Two years later he was in Oxford at work in Burden Sanderson's laboratory of physiology. In England he was thrown with such men as Michael Foster, Lauder Brunton, Klein, Gower, Stephen McKenzie and others who became his friends. In '73 he studied in Berlin under Virchow, Traube and Frerichs, and, later in the winter, he spent some months in Wien.

Sixteen years after this I had a like experience in studying in Virchow's laboratory for six months and afterwards for several months in Wien. In Berlin I took three regular university courses under Virchow; his pathological demonstrations, his course on the performing of necropsies, and his regular primary course on pathological histology. His pathological demonstrations were given in the old pathological institute in a large square room with windows on two sides. Virchow stood in the corner between the windows. In front of him the floor was covered with parallel rows of tables connected at one end so as to form a continuous line folded upon itself rather like a Greek frieze. In the middle of the tables was a track along which microscopes or plates bearing specimens might be rolled from the starting point around the entire room. The demonstrations were stimulating. Virchow showed the tissues from a series of subjects in succession, considering each case as a unit and insisting on the importance of studying the anatomical changes in a given organ in connexion with those occurring in the body as a whole. It was the complex of the changes in all the organs and their relations,

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one to another, that constituted the anatomical picture of the disease.

In performing the autopsies he removed each organ carefully and was particular as to how the gross sections should be made.

After six months in Berlin I went to Wien. There I sought immediately the pathological demonstrations at the Allgemeine Krankenhaus. These were conducted by a well-known and popular pathologist. To my amazement, there was brought before us at each demonstration a table covered with a mass of organs and tissues without relation one to another. These fragments were demonstrated separately without reference to the other organs from the same patient—an unscientific, crude and unsatisfying procedure. Moreover, the autopsies were made in a hasty and careless fashion. The organs were opened *in situ*. Again and again I have referred to the contrast between the systematic, careful procedure of Virchow and his assistants and these disappointingly slipshod methods. I cannot remember, however, to have discussed this with the "Chief."

Fancy my sensation when, many years later, I read these words from Osler's notes: "After having seen Virchow, it was absolutely painful to attend post-mortems; they are performed in so slovenly a manner and so little use is made of the material."*

Osler could have made no better beginning. In July '74 when but twenty-five years of age, he was back again in Montreal, Lecturer on the Institutes of Medicine, which in those days included physiology and pathological anatomy; at the same time he opened a consulting room. By the following spring he was full professor and Pathologist to the Montreal General Hospital. He lived in ward, autopsy room and laboratory. Cushing† and Edith Reid‡ tell of the precious records of his experience which meant so much to him and to many others in his after-life. In ten years he had become so eminent as a pathologist and a clinician that in 1885 he was called to Philadelphia as Professor of Clinical Medicine, the colleague of the distinguished William Pepper.

Four years after this, in the spring of 1889, at the age of thirty-nine, he was in Baltimore, Professor of Medicine at the Johns Hopkins University and Director of the Medical Clinic at the new hospital. It was here, in the fall of 1890, that I first came to know him. Graduating from the Harvard School in 1888 and finishing my service in the Massachusetts General Hospital in 1889, I had come back from a year in Berlin and Wien, and started my career in Boston with longing eyes turned toward the great, new venture in Baltimore; and then, what seemed to me almost a miracle, had transferred me in the twinkling of an eye, into the midst of the great experiment. It was an interesting and stimulating period, and in many ways much like what is going on here today—the opening of a new hospital, the establishment of new clinics, the introduction of new methods.

The great difference between the conditions in Baltimore in 1890 and those which had existed in the rest of the country lay in the circumstance that the medical and surgical departments were stable clinics like those of Germany, under the direction of individuals. Everywhere else the hospital services were divided between a considerable group of men whose terms occupied, as a rule, from three to four months. Often in those days there was but one house officer for a service of say fifty beds. Bacteriology was in its infancy. The clinical laboratory was a place for the examination of urine, more rarely of faeces, rather crude quantitative tests for sugar and urea, and occasional blood counts. But changes came rapidly.

They formed a fascinating group, the four chiefs at the Johns Hopkins Hospital—Osler, Welch, Halsted and Kelly. To this group Councilman, the resident pathologist, should be added. Kelly the brilliant gynecologist, Halsted the student who besides his early work on the breast and on hernia, had devised an aseptic technique more perfect than had been known before, which had transformed surgery. Welch, already recognized as the most learned of his profession, engaged in his fruitful bacteriological studies, Councilman, always stimulating, then at work with Lafleur on amoebic dysentery, and the "Chief."

I am tempted for a minute to leave my subject and to tell you of the first and strongest impression that came to me on reaching the Johns Hopkins Hospital in the fall of 1890. That first impression was made neither by Osler nor by Welch; it was made by Halsted.

I had come to Baltimore looking forward especially to the privilege of working near Welch; secondarily to the opportunity of studying under Osler. I knew little about Halsted. Coming from a hospital which rejoiced in a surgical tradition second to none in the United States, I had been familiar with good surgery, but the antiseptic technique was far from being what it is today; abdominal surgery was in its infancy. During my interneship I had never seen the abdomen opened freely save in an occasional operation for a pelvic tumour. In appendicitis one waited invariably for the formation of an abscess and attempted to open this retroperitoneally. In the three instances in which operations of this sort were done on my own patients, there was already a fatal gen-

*It should be said, however, that this procedure at the Allgemeine Krankenhaus was very different from that at Weichselbaum's laboratory in the Rudolf Spital. Neusser always followed his carefully studied cases to the dead house, and discussed them thoroughly before and during the necropsy by Weichselbaum whose methods were careful and thorough.

†Cushing, Harvey. The Life of Sir William Osler. 8°, Oxford, The Clarendon Press, 1925.

‡Reid, Edith. The Great Physician. 8°, N. Y., The Oxford Press, 1931.

eral peritonitis. Although grave post-operative infections were rare, we always waited anxiously for the end of the third day before feeling sure that the possibility of a grave infection had passed.

In the middle of October, 1890, I arrived in Baltimore. Inside of twenty-four hours I found myself in an atmosphere where the certainty of post-operative results was as assured as it is in the best clinics today; where the abdomen or the knee joint was explored without anxiety. In Halsted's little operating room, with the old wooden table, the antiseptic technique was so perfect that there was never a moment of anxiety. I could not believe my eyes. It was like stepping into a new world. At this time Halsted's technique was unique, and the sureness and perfection of his results seemed to me then and seems to me now the nearest thing to a miracle that it has been given to me to witness.

Let us come back now to the "Chief." During his fifteen years of active work as clinician and pathologist and teacher, he had never allowed his practice to eat into the time which he sought to put into study and observation, and for him the itching palm, which has destroyed so many careers, was no tempter. Sixteen years later, when he left Baltimore for Oxford, he took away not a cent more than he brought with him save the income from his work on "The Practice of Medicine."

In Montreal and Philadelphia he had acquired an experience such as was equalled by that of few men of his age. The charming simplicity of his behaviour, the thoroughness and accuracy of his procedure, and of his methods of examination, and the sureness and clarity of his reasoning impressed us deeply. Many of his house officers had been used to teachers who were more or less worshipers of authority; men to whom this or that physical sign meant something because Jones had said it did. He was familiar with the literature and he loved to refer us to the original descriptions of disease and to the newer articles of value in the current journals. He made thorough, careful examinations and he dictated rather full notes. I can see him now observing and talking to the patient, often rather facetiously, while he gained his first impression; then listening to the history; then, after his first glance, calling for a chair, sitting down and looking across chest or abdomen of his patient, with his head at the level of the body, as he pointed out to us that there are many things that one can see in this fashion which cannot be made out by other manoeuvres of eye, ear or hand—the descent of the lungs, retractions or pulsations in chest, peristaltic movements of stomach or intestines, the rounded fundus of an almost or quite impalpable gall bladder moving up and down with respiration. On his observations he reasoned as a good physiologist and pathologist. He used his ophthalmoscope constantly in the wards. The examination of the patient was not complete without the study, under an oil immersion lens, of a carefully prepared cover-glass specimen of fresh blood with which he was thoroughly familiar as a result of his important early studies on blood platelets. We rarely stained the specimens of blood in which we sought for malarial parasites, and, although I had come back full of Ehrlich's methods of blood examination, on which I worked for a large part of my first year in Baltimore, I soon came to realize that in a well made slide of fresh blood, examined immediately under the oil immersion lens, the trained observer can make out most of that which the dried specimen reveals, and a good deal which is not to be made out in the dried specimen; and further, that the examination of the fresh slide should always be the first step in the examination of the blood. Every one of us, I think, who studied under Osler, feels that a glance at a specimen of fresh blood is as vital a part of one's preliminary examination of a patient as the percussion of the chest.

Let me tell you one experience in my own practice which I have never forgotten. A woman was sent to me with the story that she had a large spleen. I found a large abdominal tumour rising apparently from the left side of the pelvis. After finishing my general examination, before making a pelvic examination, I took a cover-glass specimen of fresh blood, came back to my study, leaving the patient in the examining room, and examined the blood in the presence of her husband. While making my preparations I told the husband that I was happy to feel that the tumour was probably not of the spleen but of pelvic origin. A minute later I found myself looking at the blood of a myelogenous leukaemia. I returned, examined the woman more carefully, turned her on her right side and found that the tumour was a large, freely movable structure which had emerged from the pelvis and fallen over toward the right side of the abdomen. With a little manipulation it could be pushed back into the upper left abdominal quadrant. It had the consistency and the sharp notched border of the spleen. It was, obviously, a large, freely movable spleen which had fallen into the pelvis.

You may laugh at me for having made so gross a mistake, but I did, and examination of the slide of fresh blood saved me from an embarrassing error.

It was not only on examination of the fresh blood that he used to insist, but on the careful recording of the gross and microscopical appearances of the fresh sputa—an important procedure sometimes neglected today.

During my first year in Baltimore, Osler was at the hospital every morning and for the whole

morning. A charming and stimulating figure he was. He rarely failed to approach the patient with some bright, cheery, amusing remark singularly well adapted to the character of the individual; referring to one as a "good looking man," playfully insisting that this woman was the daughter rather than the mother, generally captivating the children by his first gesture or utterance. The wealth of his pathological experience cropped out daily and it is interesting, as one looks back, to realize how rich and well digested it was. Many clinical truths which were unrecognized by the world at large, were familiar to him on the basis of his own keen observation.

He had, for instance, become interested early in the causes of vomiting of blood, and at a meeting of the Montreal Pathological Society in 1882* he had called attention to the early and grave haematemesis in instances of splenomegaly, occurring sometimes in cases where there were no evidences of anaemia or leukaemia and at a time when the patient was in good general condition. In reply to a question from Dr. Buller, Dr. Osler remarked "that the bleeding was doubtless due in many instances to an impoverished blood stream with an enfeebled condition of the capillary walls; but in others it might be from mechanical causes. Three-fourths of the blood from the stomach is discharged into the splenic veins, and in states of engorgement of these a stasis or interference in the gastric circulation might be brought about, sufficiently intense to necessitate relief by rupture of a vessel or a diapedesis of blood into the stomach." This was a correct surmise. We boys working under him early became familiar with the grave haematemeses from dilated *vasa brevia* in splenomegaly.

He was, of course, also familiar with the relative frequency of the varicosity of the veins at the root of the oesophagus in instances of cirrhosis of the liver and of the occasional fatal haemorrhages that occur therefrom.

To the meetings of the Association of American Physicians he used to take me, as his resident physician, long before I was a member. The meeting forty years ago (in 1892) was held at the Surgeon General's Library in Washington. One of the members read a paper on a study of over two thousand instances of alcoholism. A well-known clinician opened the discussion by asking the writer if vomiting of blood were frequent in alcoholism, and related two instances of haematemesis in alcoholics occurring in his own practice. In one of these the result was fatal. As he spoke it appeared to me that he was not clear as to the possible cause, and, unconsciously, I turned my eyes toward the "Chief" whose eyes had sought mine. He preserved a proper decorum and solemnity, but winked. A few moments later he told of a like instance in his own experience of fatal haemorrhage from oesophageal varices and asked Dr. X if, in his case, there were any evidence of liver or portal engorgement and whether the oesophageal veins were enlarged*.

He followed every fatal case to necropsy and it was interesting to see the care with which he inspected the organs himself, especially the hearts. His familiarity with pathological anatomy and his wide experience leaked through, as it were, to his associates to many of whom clinical truths, not generally recognized at that time, became familiar.

All of us, for instance, were conscious of the frequency of acute endocarditis in chorea. In so admirable a work as Flint's "Practice of Medicine" in the edition of 1886, the frequency of associated endocarditis appears to be underestimated and on page 1096 one finds the following statement: "A pathological relationship is supposed to exist between chorea and rheumatism. These two affections, however, are not often associated. There is little ground for the assumption that they involve in their causation the same specific causative agent." But in 1887 Osler commented on "the extraordinary frequency with which mitral valvulitis is met with in fatal cases. *There is no known disease in which endocarditis is so constantly found, as in chorea.* * * * In a considerable proportion of cases of chorea—much larger than has been previously supposed — the complicating endocarditis lays the foundation of organic disease†."

He was among the first to recognize the clinical picture of Subacute Vegetative Endocarditis‡. I think, indeed, that to him is due the commonly accepted term applied to the clinical syndrome in English literature. In 1893 he spoke of the observations of Ormerod, Wilks, Lancereaux, Leyden, Bristowe and others, and then described a characteristic case which I well remember, a case which impressed the symptoms forever on my mind, an instance of ten months' duration. He ended by saying: "The special interest of the group illustrated by these cases is the chronic intermittent fever with progressive failure of health and strength, without dyspnoea, anasarca or the features of valvular disease." No one had put the clinical picture so clearly before the public, and his students were familiar with it for a considerable period before it became generally recognized. He had an extraordinary capacity for absorbing correctly, in an incredibly short space of time, the essence of a medical article. Little escaped him in the current medical literature. One of the rarest and most important

*Society Proceedings, Medico-Chirurgical Society of Montreal, Oct. 20, 1882. Med. News, Phila., 1882, XLI, 580-82.

*Tr. Ass. Am. Phys., Phila., 1892, VII, 67.

†Am. J. M. Sc., Phil., 1887, n. s., XCV, 371-386.

‡The chronic intermittent fever of endocarditis. Practitioner, Lond., 1893, L, 181-190.

characteristics of his teaching was the fashion in which he dwelt upon the original observations with regard to almost every interesting subject. To these articles and to every new and important contribution, he was forever referring us, but I can hardly remember his directing us to a textbook on medicine. Authority meant little to him, and those who worked beside him were taught, in studying clinical phenomena, to begin, not by comparing their results with some statement in a book, but to reason from their physiological and pathological knowledge as to what might produce such phenomena. Most textbooks of medicine are in great part, compilations. The value of Osler's remarkable work, at the birth of which we assisted, lies in the circumstance that it is based to an exceptional degree, on his own observations and experience.

In 1903 he described the disease which is sometimes known by his name, in his communication on "Chronic Cyanosis with Polycythaemia and Enlarged Spleen: A New Clinical Entity*." This clinical picture had been observed by others but by no one had it been described so clearly and so definitely as a new clinical entity. However, when the English-speaking public began to call the syndrome "Osler's disease" he promptly disclaimed his right to this distinction, and in 1908, in a paper in the *Lancet*† he calls it the "Maladie de Vaquez" who had described the condition previously—a characteristic act. But a few months ago one of Vaquez's students told me that Osler spontaneously had written a generous and cordial letter to Vaquez acknowledging his priority, a letter that Vaquez treasures as a valuable possession.

Osler taught us early that there are few more futile procedures than disputes as to priority. He who describes accurately a clinical phenomenon and gives frank recognition to everyone who has noted it before, never fails to get full credit for his work.

In Osler's early years in Baltimore he used to take a brief turn about the out-patient department after his morning visit to the wards, before going to the private wards. Later, after the school opened, he began to give, twice a week, his delightful third year clinics in the out-patient department. Interesting patients, sometimes accompanied by physicians from the outside, were brought before the class. A brief history was given by the student, or extracted in an entertaining and often racy conversation with the patient, during the exercise itself. Examining the patient before the class, Osler would express his opinion and outline the necessary studies which should be made, stopping often to comment on interesting symptoms or phenomena with regard to which he would ask the student to look up certain references and report at the next exercise. This seems to me, as I look back on it, to have been the most delightful part of his teaching. Informally and naturally, he showed his students how to approach the examination of a patient, while, at the same time, he interested them in the history of the recognition of the disease, referring them to the original descriptions of the malady and expatiating on the character and career of its discoverer. At the same time he made friends with the patient for himself and for the group. Nothing could have been more stimulating. These charming exercises led to an intimacy between him and his students which was unique, and was increased by the delightful reunions with the medical section of the fourth year class about his dining table on Saturday evenings.

Not the least valuable part of Osler's teaching was the thorough soundness of his attitude toward therapeusis. As his methods of diagnosis were simple and rational, so also were his methods of treatment.

To begin with, he recognized as did no other teacher with whom I have been associated, the importance of rest in treatment. To many of us in after years, the singular thoughtlessness with which others prescribed medicines, of one sort or another, few of them specific, without the slightest thought of the all-important physical treatment of the individual, was appalling. After living eight years in the hospital under Osler I remember my amazement at finding colleagues attempting to treat diarrhoea, for instance, where rest is nine-tenths of the affair, in patients who were allowed to go about on their feet. And we early came to recognize the vital importance of physical rest and massage in the treatment of anaemia.

At the time when I first came to Baltimore, Osler was deeply interested in malarial fever and I gave the greater part of two or three years to malarial studies. We used to hear much about the persistence of malarial fever, the difficulties of treating it, and the large doses of quinine which were necessary. Osler insisted that, so far as possible, every patient who came into the out-patient department with malarial fever should be admitted to the wards. There, in bed, it was possible to eradicate the disease much more surely and with relatively small doses of quinine. With rest in bed the ordinary tertian fevers would disappear under two grains (0.13) of quinine three times a day. But Osler never forgot the importance of increasing the dose if the patient were out of bed, and of continuing treatment at any rate for weeks and weeks. Even today there are few who realize that the ordinary malarial fevers of temperate climates may be treated and eradicated with very small doses of quinine if the patient be given complete physical rest and freedom from care.

*Am. J. M. Sc., Phila., 1903, n. s., CXXVI, 187-201.

†A clinical lecture on Erythraemia. Polycythaemia with cyanosis; Maladie de Vaquez. Lancet, Lond., 1908, I, 143-146.

"An interesting and unimportant truth," you may say; not a bit of it. Let me give you two examples.

In 1897, when reading a paper at a northern city, I was asked to see a patient who had had malarial fever for some time and who was said to be unable to take quinine. She was a woman, if I remember, in her thirties, of a sallow, anaemic, slightly yellow complexion. There was a large, palpable spleen. The blood showed a moderate number of tertian parasites in two groups. On inquiry I found that she had never been given less than ten grains of quinine at a dose, which upset her considerably. From the story it was apparent that she had not a grave hypersusceptibility to quinine, but she did feel the large doses unpleasantly. I advised that she be put to bed and kept in bed for as much as three weeks, at any rate, under two grains (0.13) of quinine every four hours; this, to be continued three times a day for several months thereafter. These small doses were taken without discomfort and she recovered completely.

One of our house officers at the Johns Hopkins Hospital had a true quinine intolerance. The slightest quantity produced urticaria and distressing symptoms. He acquired a double tertian malaria. We sent him to the mountains, put him to bed for several weeks and treated him with methylene blue, a substance which has a mild but definite anti-malarial action. In his case, with complete rest, this resulted in the complete eradication of the infection. There was no return, at least, for the subsequent four or five years during which he lived.

Nowhere, incidentally, is the information given by an immediate examination of the fresh blood, at the bedside or in the consulting room, so important diagnostically and therapeutically, as in malarial fever. There are patients who may seem at the moment to be in relatively good condition whose blood, on a glance at the fresh specimen, tells the physician that, at best, it is a race for life; that immediate intravenous treatment is necessary.

In the private wards Osler took a good many psychoneurotic patients for Weir Mitchell rest cures. No patient was ever put to bed for a rest cure without regular general massage. Not only did he use massage but he was familiar with the valuable effect of water in the form of cold and hot packs in the treatment of insomnia in nervous patients who, today, as they were then, are too often dosed with less recommendable hypnotics.

Those were the days of typhoid fever. Sometimes three-quarters of the beds in the wards were filled with patients with "typhoid". Osler realized the difference between the half-way procedures usually adopted and the well-carried-out Brand methods of bathing. All our "typhoids" were treated by regular tub baths, repeated usually at intervals of three hours if the temperature were above 102.5°. There were movable tubs in every ward. Canvas strips were clamped with brass clips to the rims of the tub so that the back was supported and the patient was relieved from undue strain. The nurses and orderlies were taught properly to rub the patient while in the bath. The results were remarkably good. Those of us who are familiar with the superiority of bathing to the half-way methods of using water then popular, can but lament its frequent abandonment for the old, sloppy procedures of forty-five years ago.

He taught us the value, in general, of physical methods, and he taught us reason and moderation in therapy.

In the use of opium, mercury and iodide of potassium, of quinine and of digitalis, he was wise, careful and exact. He was inclined to use arsenic in Hodgkins disease and in pernicious anaemia and, of course, in leukaemia. He never meddled with drugs unnecessarily in the treatment of his pneumonias. He was moderate but judicious in the use of alcohol. He never gave a patient internal treatment without a perfectly good reason.

When the medical school opened he directed his attention especially to the care of the more important diseases—syphilis, tuberculosis, typhoid fever, pneumonia and the anaemias. The course of the pneumonias and typhoids of the year was tabulated week by week, and remained on the blackboard in the main lecture room throughout the year. Every student in the third and fourth years left the school with a personal experience in typhoid fever and pneumonia—that of a good-sized hospital service of two years' duration.

In public and in private he waged an unceasing campaign in favour of the prevention, the early recognition and the proper care of typhoid fever and tuberculosis.

With the exception of the circumstance that he was a little slow to see the value of feeding in typhoid fever, his teaching as to the treatment of this disease was a model. Especially did he devote himself to denouncing the public scandal of the prevalence of the disease, and the unfortunate confusion in the minds of the profession as to its nature and relations to an entirely different and easily recognizable process, such as malarial fever. It is hard to realize today the conditions which existed in the early nineties—the lack of information of the profession at large with regard to the nature of typhoid fever, and the wholesale and un-understanding use of the word "malaria". This word was forbidden in the medical clinic of the Johns Hopkins Hospital. The diagnosis "Malarial fever" was made when the nature of the disease was proven, but Osler never tired of insisting before students and colleagues that the word "malaria," as used in this country, was as vague and meaningless as the expression "biliousness"

which has a different meaning to every individual who uses it, and no meaning in itself.

A worthy but rather ill-educated physician, regarding typhoid fever as a local infection of the gastro-intestinal tract in the sense that the intestines were full of typhoid bacilli, and that all that was needed properly to treat it was a method of intestinal antisepsis, which he innocently felt was a relatively simple matter, evolved a meddlesome and sometimes a dangerous method of treatment involving considerable purgation and the almost hourly administration of medicines of one sort or another. Without the advantages of association with a large hospital, with small means of confirming his diagnoses, unable properly to recognize the disease, he asserted repeatedly that no man need die of typhoid fever, and that he had discovered a form of treatment which was almost specific. One of the best-known firms of manufacturing chemists in the country produced and advertised the necessary equipment for "X's method" of treatment of typhoid fever, and sent agents far and wide over the United States to urge its use.

In the fall of 1899 I attended a meeting of the Seaboard Medical Society in Weldon, N. C. This firm had sent a representative to the meeting. He was a physician who had retired from practice, if indeed he had ever practised. His occupation, at that time, appeared to be making campaign speeches for whichever party desired him, during the political campaigns in the fall, and extolling the value of "X's method" of treatment of typhoid fever and selling or advertising the equipment for this throughout the country.

I happened, at this meeting, to be speaking on the nature of typhoid fever and its treatment, and I discussed at length what one might reasonably expect to attain by the therapeutic methods available. The agent did not speak at the meeting before which I appeared, but after the morning session, a considerable group of members were invited to his room where he opened several bottles of champagne. He was a genial fellow and made an agreeable host. I was unable to stay for the dinner that evening. Before I left one of the members said: "I am so sorry you can't stay for the dinner;——, our friend the agent, is going to speak. He is an awfully good speaker, and he is going to give you the devil." I have always been sorry that I might not have heard what he said, if he said it. This, however, I know; that I have never, from that day, bought a product of the laboratory of this firm if I could possibly avoid it.

No one will ever know how many lives may have been lost by this meaningless procedure against which Osler spoke on every possible occasion. When he spoke there was no doubt as to his meaning. In the address in general medicine delivered in 1896 at Atlanta* he said: "* * * As a strong advocate of hydrotherapy, I take especial pleasure in denouncing as heretics of the worst possible stamp, the advocates of the so-called antiseptic and abortive methods of the treatment of typhoid fever. I would place the man who does not for this purpose also give a purge, in a limbo just a little less hot. * * *"

*The study of the fevers of the South. J. A. M. A., Chicago, 1896, XXVI, 999-1004.

I have spoken of the intimacy of Osler's relations with his students, and everyone who has written about him has told of the Saturday evening gatherings of the group of fourth year men who were doing their work in the medical wards. The medical group of the fourth year students of the first class used to meet me on alternate Tuesday evenings, but when the second class came along Dr. Osler invited them weekly to his house. Soon after dinner they gathered around his dining table, he at the head, to discuss the problems that had come up in the wards, but especially to listen to the "Chief's" delightful conversation.

During those evenings, over beer and lemonade and sandwiches, the boys learned much of the history of medicine, of books and of the biography of the great men who had gone before; but more than all they came to know and love the "Chief" and the "Chief" came to know them. The "Chief" really knew well every student who went through the school.

His relations with the nurses were likewise charming. Every physician who has been connected with a hospital knows of the occasional complications that arise in connexion with the training school. Many men, until they have acquired the necessary experience, fail to regard as they must, the more delicate sensitiveness of women who cannot always be treated with the same brusqueness and crudity with which men treat one another. There were few difficulties when it came to Osler. He was so imperturbably bright and cheerful, so light of touch in dealing with everybody. His sense of humour was always in play, and, fundamentally, he was so considerate of everyone, that there was never a quarrel or a difficulty in his dealings with men or women. He had an almost miraculous power of turning aside wrath. It was difficult—nay, impossible—to involve him in a discussion which he perceived would be futile; and his powers of perception were quick and sure. The miracle of it all was, that, although the baffled and discontented complainer might part from him somewhat vexed, it was almost invariably with a smile and rarely with resentment.

Much has been written about his gaiety and his love of little—someone has called them "impish"—practical jokes, which were never malicious. He went through life with a song, whistling by the way, as he said, "* * * that I may not weep." His relations with his colleagues were altogether charming. With all his sense of humour and the eternal twinkle in his

eye, he never said an unkind or a slighting word of a colleague. As Edith Reid has said, every man knew that his reputation was safe with Osler. If he could say no good of a man he said nothing at all, and gossip of any sort was impossible in his presence. In his consultations and his professional relations he was direct and truthful, infinitely tactful and kindly, and always helpful. He was, to an extraordinary degree, master of his own time, and he had an art of guarding his own time which I have never seen equalled.

He was the most charitable and kind-hearted of men, almost unreasoning, sometimes, in his prejudice against the coldness of organized charity. He never refused a beggar.

Into the depths of feeling which lay behind and beneath his outward gaiety no one, unless it was his wife, ever penetrated. He used to speak of the virtue of taciturnity. There are some things of which no wise man ever speaks.

One lived and worked alongside of Osler in an atmosphere of healthy and happy enthusiasm. A week rarely passed without the addition of some bright, amusing remark, some new epigram or some charming and characteristic incident to the store which we treasured. We loved him and respected him deeply but, treated as companions rather than as students or disciples, we hardly realized how closely he had entered into our lives. I had always expected that he would end his days in the Old Country. I had known how much tempted he had been to accept a call to Edinburgh. I should have fancied that the announcement that he was going to Oxford, while filling me with regret, yet would have come as a piece of news more or less to be expected. When the announcement did come I was hiding in a Washington hotel for several days in order that I might give my time uninterruptedly to some writing. As I read the announcement in the evening paper I was completely overcome. I could think of nothing else, and, despite myself, I lay down on the bed and wept like a child. Others must have had a like experience. I had no idea until that moment, of the part that he had played in my life.

I have not even mentioned Lady Osler—"Mrs. Chief," as we used to call her lovingly—a beautiful and remarkable woman who played an inestimable part in the happiness and success of his life. We who knew them cannot think of them apart.

Again, I have not spoken of his son, the great hope of his life, whose death—he was killed in the war—was its crowning tragedy. Some day when you are in Baltimore ask a member to show you the rooms of the Tudor and Stuart Club. Revere Osler, whose mother was a great-granddaughter of Paul Revere, had become an enthusiastic collector of books. This collection, along with some of his own treasures, Osler gave as a part of the foundation of a literary club for the students of the Johns Hopkins University, a precious memorial.

At Oxford he found rest from demands which were gradually overwhelming him, few and light duties from the standpoint of teaching, and an unparalleled opportunity to indulge himself in his bibliographical avocations. He was received with open arms by his British colleagues and, although he rather shunned practice, his advice was widely sought in public and in private, and his preëminence was universally recognized. A Student of Christ Church, he became a dearly loved member of the great family of scholars in and about the old University. As in Baltimore, his house had become almost the home of his students and friends, so in Oxford, 13, Norham Gardens became known among his friends, as the "Open Arms." In the afternoons his parlour and garden were the happy gathering places of friends and students, especially Rhodes scholars. In the last year of his life, over sixteen hundred guests sat at his dining table.

The war was a sad trial in many ways. He threw himself into the work whole-heartedly. He did far more than his part, and he made his supreme contribution without a murmur. When the war was over he was the first to go to the aid of the sufferings of those who but yesterday had been his enemies.

He was as dearly loved in England as he was with us; England was his home and his countrymen welcomed him with generous pride as a compatriot and joined with his friends of the New World in honouring his memory.

I fancy that some of the companions of Osler in his earlier decades hardly conceived, at the time, of the impress which their very human and disconcertingly spirited associate was to leave upon the world. They would perhaps have smiled had they been told that after his death, many would look up to Osler with an adoration almost equal to that which is bestowed on a saint. "A very unorthodox saint!" some would have said. But remember that all saints were once human. A saint before the age of forty is a pathological and a rather unpleasant phenomenon. Only when one has passed through the fire and trial of temptation which every high-spirited man must know; only when one has erred and suffered can he attain that understanding and charity and tolerance and serenity and equanimity which set him apart from other men. Only after death does one's life stand out in full relief. Only when one is dead does he become a saint.

I like to think that the ivy which we have planted today will keep ever young these halls, in the memory of a great physician and of a truly great and inspiring character, one who loved his fellow men; one who is already canonized in the hearts of those who surrounded him on earth. For to us who were close to him during the last thirty years of his life, he will always stand out a figure apart from most other men.

A SERIES OF CASES PRESENTING SYNDROMES SUGGESTING POLIOMYELITIS*

BY DAVID W. SHERWOOD, M.D.†

DURING any epidemic of poliomyelitis various disease entities are mistaken for poliomyelitis. Aycock, Kramer, and others reported a series of such cases in 1929[1]. While working with the Harvard Infantile Paralysis Commission in 1931 it was my privilege to observe syndromes which simulated poliomyelitis by certain signs and symptoms but which proved to have a different etiology.

The majority of cases reported in this paper were seen in 1931. During that year approximately 1211 cases were seen by the Commission. About 750 of these cases were poliomyelitis. The remainder formed the groups from which the majority of cases reported in this paper were selected. A few were observed in 1928. All cases were seen at the request of physicians as questionable poliomyelitis. Each has been carefully followed to establish a final diagnosis where the cause of the illness was not evident at the first visit. A typical case of each disease entity seen as questionable poliomyelitis has been selected. There were many duplications, especially of upper respiratory infections.

An attempt is made to point out signs and symptoms and laboratory data which aided in making a differential diagnosis. A brief history of the positive findings is given in each case. Negative findings are mentioned only when they are significant in respect to the diagnosis.

The signs and symptoms of poliomyelitis have been described many times in the literature. I shall attempt to review only the cardinal signs and symptoms in the typical case.

The disease in its preparalytic and postparalytic stages is most frequently noted in the late summer and early fall. The young are most susceptible and the disease most often occurs between the ages of one and five, although there is some variation in the age distribution from year to year. The children who are stricken with poliomyelitis appear to present a definite type. They are usually bright, alert, and coöperative. They frequently have round heads and full faces and seem to have been in a state of excellent health previous to the attack of the disease.

Clinically the preparalytic stage of this disease can progress into two types, namely, spinal and bulbar. There may be a combination of these two types. An encephalitic type has been described. This is very rare, if it actually does occur, and the literature is not in agreement concerning the etiology of the disease which has been described as "polioencephalitis".[2]

The onset is usually abrupt in both types. It may be preceded in the dromedary cases by a previous illness, usually occurring five days before the actual onset of the disease, consisting of fever, headache, and vomiting. Perhaps only one of these symptoms will be noted, or a combination of two of them may be observed. At this time the spinal fluid is negative but becomes positive at the actual onset of the disease, or at the onset of the second fever in the dromedary type. In the interim the patient is well. Not all cases present the dromedary phase.

In the spinal type the disease is usually ushered in with a temperature ranging from 100 to 102½ degrees. Headache and vomiting are present in the typical case. Constipation is a frequent symptom. Diarrhea is rare. Muscle tenderness occurs infrequently. Pain on motion is most noticeable with respect to the neck and spine. The patient is rational and coöperates with the doctor during the examination. Prostration in the true sense of the word (to reduce to extreme weakness, great dejection, or depression) does not seem to be present. The patient may appear drowsy when left alone but is alert when examined. A slight peritonsillar injection is often noted, but the throat does not appear to be severely enough injected to be the cause of the symptoms. Tremor is frequently present. Stiff neck and spine are almost constant signs although the stiffness may not be marked. It will be found that the patient is unable to flex his chin to his chest or to bend his chin to his knees while in a sitting position without evidence of splinting and pain. A tache cérébrale is observed in the majority of patients. Hyperesthesia was not a frequent finding in 1931. The reflexes may be moderately increased or absent. The white blood count may be from 14,000 to 20,000. The spinal fluid findings show increased pressure, clear or ground glass fluid, positive globulin, increased cell count, normal sugar and increased total protein. The cell count in the spinal type is likely to be higher than in the bulbar.

The bulbar type of the disease may present all the signs and symptoms mentioned in the spinal type or, more rarely, it may present no symptoms but may first be noted with the appearance of bulbar involvement causing paralysis. The temperature may be as high as 104 or 105 degrees in the typical case. The pulse, too, may be 150 or 160 or there may be a bradycardia. The breathing is usually accelerated. Dysphagia is a prominent symptom. This type of the dis-

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ease may be much more fulminating than the spinal type and death may occur in a short time due to the involvement of the higher centers. The spinal fluid cell count as mentioned above may be low in the bulbar type. A cell count of ten or more combined with the other spinal fluid findings and the clinical picture described above is compatible with this type of the disease. Cell counts of less than ten have been reported in certain cases. In the spinal type the cell count may range from 50 to 200 and occasionally as high as 2000. Early in the disease polymorphonuclear leucocytes often predominate in the spinal fluid, but later the lymphocyte is usually the predominating cell.

The paralytic stage of the disease is the end result of the preparalytic syndrome. That paralysis is not a necessary sequela of the preparalytic syndrome is well known. The paralysis when it does occur is flaccid. Any muscle may be affected. A spastic type of paralysis has been described in the cerebral type but, as mentioned above, data to prove that the virus of poliomyelitis is the agent concerned are not convincing at the present time. Paralysis usually develops about the third or fourth day after the onset of the disease. When paralysis occurs the acute stage may be at its height, subsiding or practically over. The paralysis may be attended by acute muscle pain, and this pain may continue well into the convalescent stage of the disease.

The cases reported in this paper have been divided into two main groups.

Group I contains those cases which presented meningeal signs or signs simulating them. This group has been divided into three sub groups, A, B, and C.

Sub group A consists of cases in which the spinal fluid findings showed a pathological process to be present. Sub group B consists of cases which showed a true meningism with no change in the spinal fluid indicative of pathology. Sub group C consists of those cases in which the signs present were mistaken for meningeal signs.

Group II contains those cases which presented actual paralysis or paresis or muscular involvement simulating paralysis. This group has been divided into two sub groups, D and E.

Sub group D contains those cases which presented true paralysis or paresis which were due to etiology other than that of poliomyelitis. Sub group E consists of those cases which had muscular involvement which was mistaken for paralysis.

Group I

Cases presenting meningeal signs or signs simulating them.

Sub group A.—Cases showing pathological spinal fluid findings.

1. Brain Abscess
2. Encephalitis
3. Meningitis-atypical paratyphoid bacillus
4. Meningitis—mumps
5. Meningitis—tuberculosis
6. Meningitis—meningococcus
7. Meningitis—influenza
8. Meningitis—postoperative
9. Meningitis—pneumococcus
10. Syphilis

Sub group B.—Cases with meningism.

11. Asthma
12 and 13. Cholangitis
14. Dysentery
15. Measles
16. Osteomyelitis
17. Pityriasis
18. Pneumonia
19. Pyuria
20. Rheumatic State
21. Serum Disease
22. Trichinosis
23. Upper Respiratory Infection.
24. Varicella

Sub group C.—Cases with signs simulating meningeal signs.

25. Appendicitis
26. Cervical Abscess
27. Hysteria
28. Tetany
29. Tetanus

Group II

Cases with Paralysis, Paresis or signs simulating Paralysis.

Sub group D.—Cases with Paralysis or Paresis.

30. Bell's Palsy
31. Cerebral Hemorrhage
32. Chorea
33. Cord Tumor
34. Diphtheria
35. Epidural Abscess
36. Polyneuritis
37. Tuberculosis—miliary
38. Tuberculosis of the spine
39. von Recklinghausen's Disease

Sub group E.—Cases with signs simulating paralysis.

40. Epiphysitis
41. Laryngitis
42. Osteomyelitis
43. Scurvy

The cases in Group A caused poliomyelitis to be considered as a differential diagnosis because a true stiff neck and spine were present. There were seven cases of meningitis in this group. Five of these seven cases of meningitis had other signs and symptoms present, such as a fever of

105° without bulbar involvement, coma, irrational state, marked prostration or a history which should have led one to suspect meningeal involvement other than that due to poliomyelitis. Mumps meningitis and meningococcus meningitis needed further investigation and observation in order to eliminate poliomyelitis as a possible diagnosis. Cases of brain abscess and encephalitis are included in this group. They presented signs of coma and an irrational state which, combined with the history and other physical findings, should have suggested cerebral involvement. Syphilis in infancy is also included in this group. There were local signs, such as joint swelling, which were incompatible with poliomyelitis.

As mentioned above, the spinal fluid findings in these cases showed a pathological process to be present. Brain abscess, encephalitis, syphilis and mumps meningitis showed spinal fluid findings which were compatible with the usual findings in the spinal fluid of poliomyelitis. Mumps meningitis showed a faintly cloudy fluid. Poliomyelitis shows a clear or ground glass fluid. That this difference in the appearance of the fluid was of importance in this case was proved the second day of the disease when a diagnosis of mumps meningitis was made because of the swelling of the parotid glands. In these cases the other signs and symptoms mentioned above were helpful in excluding poliomyelitis as a diagnosis. The other cases in this group showed spinal fluid changes which were typical of other disease but not of poliomyelitis.

In Group A, a lumbar puncture was needed as a diagnostic aid to rule out poliomyelitis in two cases, namely those of mumps and meningococcus meningitis. In the other cases the history, signs, and symptoms presented sufficient evidence to eliminate poliomyelitis as a diagnosis but not sufficient evidence, perhaps, to make a final diagnosis.

Group B consists of fourteen cases of different disease entities showing meningism with acute infection. Meningism is understood to mean a condition of irritation to the brain or spinal cord in which the symptoms simulate meningitis, but in which no actual inflammation of these membranes is present. Globulin is sometimes present in the spinal fluid in this condition.

Poliomyelitis was considered as a differential diagnosis in this group because the cases presented a stiff neck and spine. In twelve of the fourteen cases the history, combined with the signs and symptoms, presented sufficient evidence to establish a diagnosis without the diagnostic aid of a lumbar puncture. In seven of these twelve cases, however, a lumbar puncture was done to dissipate any fear, which arises in the minds of all of us during an epidemic, that the disease present might, even in the presence of the signs and symptoms of other disease entities, after all be poliomyelitis. The spinal fluid findings in these seven cases were negative. In two cases which later proved to be cholangitis a lumbar puncture was done because poliomyelitis could not be ruled out without this diagnostic aid. The spinal fluid findings proved negative in these two cases and typical cases of cholangitis developed.

In these fourteen cases meningism was present. This was proved in nine cases by negative spinal fluid findings and by the course of the other five cases. One exception may be taken to this statement as the case of osteomyelitis died and no autopsy was done to prove the absence of meningeal involvement. The clinical diagnosis was osteomyelitis and septicemia, however.

In Group C there were five cases which presented signs due to muscular splinting or irritation which were mistaken for meningeal signs.

A case of appendicitis had a stiff back which was a protective mechanism for the abdominal pain. The cervical abscess case presented a stiff neck and spine as a protection against pain caused by the abscess. In the case of hysteria, a lumbar puncture was done which showed a negative spinal fluid. This was not a case of true meningism as the course of the illness proved. In the cases of tetany and tetanus there was true muscular spasm which suggested meningeal irritation.

None of these cases resembled poliomyelitis in respect to signs or symptoms, but the muscular involvement suggested meningeal irritation which in turn suggested poliomyelitis.

Group D, under the heading of Group II, contains those cases which presented true paralysis or paresis.

Cases of Bell's palsy, diphtheria, epidural abscess, and polyneuritis in this group showed a flaccid paralysis similar to that found in poliomyelitis. The history, signs, and symptoms gave sufficient evidence to eliminate poliomyelitis as a diagnosis except in the case of Bell's palsy where a lumbar puncture was done. This proved to be negative and was considered to be indicative of the absence of poliomyelitis.

Cerebral hemorrhage, cord tumor, and tuberculosis of the spine showed evidence of upper motor neuron involvement. The case of cerebral hemorrhage showed spasticity and the other two cases showed paralysis with the reflexes intact. None of these findings are compatible with poliomyelitis which shows a flaccid paralysis with lower motor neuron involvement.

Cases of chorea and von Recklinghausen's disease presented signs of paralysis or paresis in the presence of other cerebral signs. A case of miliary tuberculosis presented paralysis which is often noted in this disease when there is meningeal involvement. Other signs of miliary tuberculosis were present to help establish a diagnosis. In this case, as in Case 5, which was tu-

berculous meningitis, the spinal fluid findings differed from those of poliomyelitis only in regard to the sugar. The sugar in the spinal fluid is usually reduced to the qualitative test in tuberculous meningitis while it is normal in poliomyelitis. This is often an important differential point.

Lumbar punctures were done in five of these ten cases. In Bell's palsy, as mentioned above, it was necessary to prove or disprove the existence of poliomyelitis. In the other four cases, namely, cerebral hemorrhage, cord tumor, epidural abscess, and miliary tuberculosis, a lumbar puncture was done to prove the existence of disease other than poliomyelitis. The spinal fluid findings in the case of epidural abscess were compatible with poliomyelitis, but the signs and symptoms were not those of poliomyelitis.

In ten cases presenting paralysis, poliomyelitis could be eliminated as a diagnosis in nine by means of the history, physical signs, and symptoms. Further investigation was necessary in some of the nine cases, however, before a final diagnosis could be made.

Group E contains four cases showing muscular involvement which was mistaken for paralysis. The case of acute laryngitis was suspected of having bulbar poliomyelitis with intercostal involvement because of the dyspnea. Cases of epiphysitis, scurvy, and osteomyelitis showed muscular splinting which was thought to be paralysis. The splinting was present as a protective mechanism to prevent pain which was caused by the disease processes present. No lumbar punctures were done on the cases in this group.

SUMMARY

Forty-three cases of different diseases which were suspected of being poliomyelitis are reported.

Each case presented some symptoms which were somewhat suggestive of poliomyelitis. In eighteen cases the diagnosis was established without the aid of a lumbar puncture. In these cases the clinical signs and symptoms were conclusive evidence that a process other than poliomyelitis was present. In twenty-five cases a lumbar puncture was done. In five of these twenty-five cases it would have been impossible to rule out poliomyelitis without this diagnostic aid. These were cases 4, 6, 12, 13, and 30. In the remaining twenty lumbar puncture cases a specimen of the spinal fluid was taken to prove the existence of a disease other than poliomyelitis, or to dispel the fear that poliomyelitis might be present even in the absence of the typical signs and symptoms. That is, in these cases there was some suggestion from the signs and symptoms or from the course of the disease that would lead one to suspect he was not dealing with poliomyelitis. The cases requiring a lumbar puncture in the absence of the typical signs and symptoms of poliomyelitis were cases 1, 2, 3, 5, 7, 8, 9, 10, 11, 15, 17, 18, 19, 22, 23, 27, 31, 33, 35, 37.

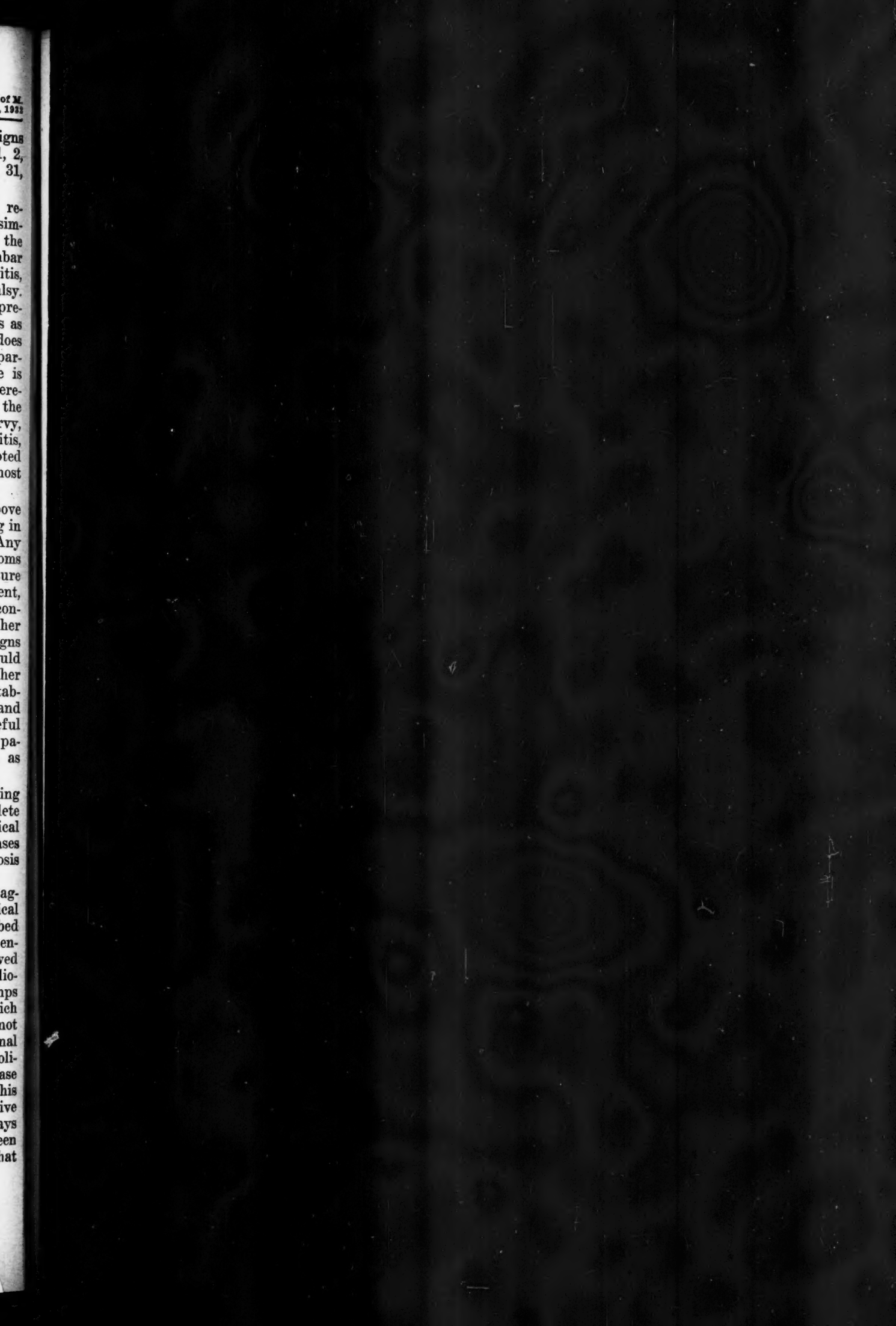
Therefore five of the forty-three cases reported presented signs and symptoms which simulated poliomyelitis closely enough to leave the diagnosis in doubt without the aid of a lumbar puncture. Two of these cases were meningitis, two were cholangitis and one was Bell's palsy.

Poliomyelitis in the preparalytic stage presents a definite clinical picture in most cases as described above. The paralysis when it does occur is a characteristic lower motor neuron paralysis. The reflex is gone and the muscle is flaccid. Muscle involvement in neuritis, cerebral hemorrhage, diphtheria, pressure on the spinal cord due to abscess or tumor, scurvy, hysteria, syphilis, tuberculosis, osteomyelitis, rheumatic state, and serum sickness are noted above. The type of muscle involvement in most of these cases did not suggest poliomyelitis.

It will be noted from the cases described above that any of the signs and symptoms occurring in poliomyelitis may occur in other diseases. Any deviation from the typical signs and symptoms of poliomyelitis, however, such as a temperature of 104° to 106° without bulbar involvement, coma, irrational state, marked prostration, convulsions or the signs and symptoms of other definite syndromes with only a few of the signs and symptoms of poliomyelitis present should lead one to look for a cause of the illness other than poliomyelitis. In addition, when establishing a differential diagnosis, the history and the course of the disease should receive careful consideration. The age and the type of the patient are helpful, but of minor importance as evidence of the presence of poliomyelitis.

Too much emphasis cannot be laid on taking a meticulous history and making a complete physical examination with careful neurological observations. In eighteen of the forty-three cases reported, as mentioned above, a final diagnosis was made by this method.

The spinal fluid in poliomyelitis is not diagnostic of the disease in the absence of the typical signs and symptoms. In the cases described above, brain abscess, encephalitis, mumps meningitis, syphilis, and epidural abscess showed fluid similar to the spinal fluid findings in poliomyelitis. Each of these diseases except mumps meningitis showed signs and symptoms which should have led one to suspect that he was not dealing with poliomyelitis. A negative spinal fluid, however, in the presence of suspected poliomyelitis is good evidence against the disease unless found in the dromedary stage. At this time it will be negative only to become positive at the actual onset of the disease several days later. Negative spinal fluid findings have been reported in poliomyelitis but are so rare that



	Diagnosis	Age	Sex	Fever	Vomiting	Headache	Convulsions	State of Mind	Prostration	Constipation	Diarrhea	Stiff Neck	Stiff Spine	Tremor	R
						SYMPTOMS, PHYSICAL SIGNS AND									
Group I—*Subgroup A.* (Pathological spinal fluid findings.)															
Case 1	Brain Abscess	27 yr.	Male	Moderate	Yes	Yes	No	Coma Irrational	Yes	—	No	Yes	Yes	—	
" 2	Encephalitis	5 yr.	"	103	"	"	"	Coma Irrational	"	Yes	No	"	"	—	Inc
" 3	Atypical Paratyphoid Meningitis	8 yr.	"	105	"	"	"	Irrational	"	—	—	"	"	Yes	
" 4	Mumps Meningitis	7 yr.	"	Yes	"	"	"	—	No	—	—	"	"	—	
" 5	TBC Meningitis	18 mo.	Female	Low	"	"	Yes	Coma Irrational	Yes	—	—	"	"	Yes	Inc
" 6	Meningococcus Meningitis	17 yr.	Male	100.6	"	"	No	Clear	No	No	No	"	"	—	
" 7	Influenza Meningitis	6 yr.	"	Moderate	"	"	"	Irrational	Yes	—	—	"	"	Yes	
" 8	Postoperative Meningitis	16 yr.	"	103	"	"	"	Coma	"	—	—	"	"	—	
" 9	Pneumococcus Meningitis	4 yr.	Female	Yes	—	—	—	Coma	"	—	—	"	"	—	
" 10	Syphilis	2 mo.	"	100	Yes	—	—	No	No	—	—	"	No	—	No
Group I—*Subgroup B.* (Meningism.)															
Case 11	Asthma	22 yr.	Female	Yes	No	Yes	No	Clear	No	—	—	Yes	Yes	—	Inc
" 12	Cholangitis	21 yr.	Male Brother of Case 13	Yes	Yes	"	"	Clear	No	No	Yes	"	"	—	
" 13	"	23 yr.	Male Brother of Case 12	Yes	"	"	"	"	"	"	"	"	"	"	—
" 14	Dysentery	5 yr.	Female	104	"	"	Yes	"	"	"	"	"	"	"	—
" 15	Measles	2½ yr.	"	106	"	"	"	Semi-comatose	Yes	—	No	Yes	No	Yes	Inc
" 16	Osteomyelitis	11 yr.	"	104	"	"	No	Clear	No	No	"	"	Yes	—	
" 17	Pityriasis Rosea	9 yr.	Male	102	"	"	"	Clear	"	"	"	"	"	Yes	
" 18	Pneumonia	6 yr.	"	104	"	"	"	"	"	"	"	"	"	—	
" 19	Pyuria	10 yr.	Female	102	"	"	"	"	Yes	—	—	No	"	—	
" 20	Rheumatic State	6 yr.	Male	102	"	"	"	"	"	Yes	No	Yes	"	—	
" 21	Serum Disease	7 yr.	"	101.6	No	No	"	"	"	No	"	"	"	—	Incr
" 22	Trichinosis	19 yr.	Female	101	"	Yes	"	"	No	Yes	"	"	"	—	
" 23	Upper Respiratory Infection	1[illegible] yr.	Male	104.4	"	"	"	"	"	"	"	"	"	Yes	
" 24	Varicella	13 yr.	"	Moderate	"	"	"	"	"	"	"	"	"	—	
Group I—*Subgroup C.* (Signs simulating meningeal signs.)															
Case 25	Appendicitis	2 yr.	Female	101	Yes	No	No	Clear	Yes	No	No	No	Yes	Yes	
" 26	Cervical Abscess	6 yr.	Male	102	"	"	"	"	No	—	No	Yes	"	No	Norm
" 27	Hysteria	21 yr.	Female	No	No	"	"	"	"	No	No	No	"	—	Prese
" 28	Tetany	17 mo.	Male	"	"	"	Yes	—	—	—	—	"	No	Yes	Incr
" 29	Tetanus	5 yr.	Female	"	"	"	No	Clear	Yes	—	—	Yes	Yes	"	

SE REPORTS

NS AND FACTS PERTAINING TO CASES

rem-or	Reflexes	Paralysis	Spinal Fluid					History	Miscellaneous
			Character	Globulin	Sugar	Cells	Org.		
—	—	No	Pressure +	+	Normal	85	—	—	—
—	Increased	No	"	"	"	80	—	Vaccinated 13 days previous to P.I.	Pain in abdomen Kernig + Babinski +
Yes	—	No	"	"	—	2300	Atypical paratyphoid bacillus	—	—
—	—	No	Cloudy	"	—	980	—	Paratoid swelling following day	
Yes	Increased	Yes	Pressure +	"	Reduced	140	—	Sick 17 days tbc in family	Rt. patella clonus
—	"	No	Cloudy Pressure +	"	—	1600	Meningococcus	—	Kernig +
Yes	"	No	Cloudy Pressure +	"	—	—	Influenza	—	"
—	"	No	Cloudy Pressure +	"	—	2500	—	Mastoidectomy 1 week previously	Babinski + Kernig +
—	"	No	Cloudy Pressure +	"	Decreased	1100	Pneumo Type 2	—	Pupils failed to react to light
—	No	Pseudo	Clear	"	Normal	30	—	Joint swelling	X-ray showed bone destruction Blood Wass. +
—	Increased	No	Clear	Neg.	—	0	—	Responded to asthma therapy	Rapid respirations
—	"	No	"	"	Normal	0	—	Jaundiced on following day	Pain in back Kernig +
—	"	"	"	"	"	0	—	"	"
—	"	"	—	—	—	—	—		Stool showed blood and mucus
Yes	Increased	No	Clear	Neg.	—	0	—	—	Morbilliform rash
—	"	"	—	—	—	—	—	—	Swelling over rt. femur Kernig +
Yes	"	"	Clear	Neg.	Normal	0	Culture neg.	—	Branny macular rash
—	—	"	"	"	—	0	—	—	Dilated alae nasi Resp. 45 Pulse 140 X-ray showed pneumonia
—	—	"	"	"	—	0	—	—	Costovertebral tenderness Urine showed pus
—	—	"	—	—	—	—	—	—	Red, swollen joints Dyspnea
—	Increased	"	—	—	—	—	—	9 days previously R antitetanus serum	Urticarial rash
—	"	"	Clear	Neg.	Normal	0	—	Ate raw pork	Edema under eyes Eosinophilia
Yes	"	"	"	"	"	0	—	Proved by course of the disease	Infected throat Pain in legs and back
—	—	"	—	—	—	—	—		Papulovesicular rash
Yes	—	No	—	—	—	—	—		Abdominal pain, spasm and tenderness
No	Normal	"	—	—	—	—	—		Tenderness and swelling cervical spine
—	Present	"	Clear	Neg.	Normal	0	—	Proved by course of the disease	Hyperesthesia Weakness of legs
Yes	Increased	"	—	—	—	—	—		Rachitic Rosary Carpopedal spasm
"	"	"	—	—	—	—	—	Vaccinated 5 days	Opisthotonos

GROUP I—*Subgroup C.* (Signs simulating meningeal signs.)																
Case 25	Appendicitis	2 yr.	Female	101	Yes	No	No	Clear	Yes	No	No	No	Yes	Yes	—	
" 26	Cervical Abscess	6 yr.	Male	102	"	"	"	"	No	—	No	Yes	"	No	Normal	
" 27	Hysteria	21 yr.	Female	No	No	"	"	"	"	No	No	No	"	—	Present	
" 28	Tetany	17 mo.	Male	"	"	"	Yes	—	—	—	—	"	No	Yes	Increased	
" 29	Tetanus	5 yr.	Female	"	"	"	No	Clear	Yes	—	—	Yes	Yes	"	"	
GROUP II—*Subgroup D.* (With paralysis or paresis.)																
Case 30	Bell's Palsy	3½ yr.	Male	101	Yes	Yes	No	Drowsy	No	No	No	No	No	—	—	
" 31	Cerebral Hemorrhage	3 yr.	Female	No	No	No	"	Unconscious	Yes	—	—	"	"	—	Active	
" 32	Chorea	16 yr.	"	Moderate	"	"	"	—	—	—	—	"	"	—	—	
" 33	Cord Tumor	56 yr.	"	No	"	"	"	—	—	—	—	"	"	—	Active	
" 34	Diphtheria	5 yr.	"	"	Yes	"	"	—	Yes	—	—	"	"	—	K. J. Absent	
" 35	Epidural Abscess	16 yr.	"	104	—	—	"	Clear	—	—	—	"	Yes	—	K. J. Absent	
" 36	Polyneuritis	42 yr.	Male	No	No	No	"	"	No	—	—	"	No	Yes	—	
" 37	Miliary Tuberculosis	17 mo.	Female	101	Yes	—	Yes	Stupor	Yes	Yes	—	Yes	Yes	"	—	
" 38	Tuberculosis of the Spine	7 yr.	Male	No	No	No	No	Clear	No	"	—	No	"	—	Present	
" 39	von Recklinghausen's Disease	10 yr.	Female	"	—	—	—	"	"	—	—	"	No	Yes	Abnormal	
GROUP II—*Subgroup E.* (Signs simulating paralysis.)																
Case 40	Epiphysitis	13 yr.	Male	102	Yes	Yes	No	Clear	Yes	—	—	No	No	—	—	
" 41	Laryngitis—Acute	4 yr.	"	102	"	"	"	"	No	—	—	"	"	No	Normal	
" 42	[illegible]omyelitis	3 yr.	"	102	"	—	Yes	—	Yes	—	—	"	"	—	—	
" 43	Scurvy	6 mo.	"	No	No	—	No	Clear	No	—	—	"	"	—	Present	

—	No	—	—	—	—	—		Abdominal pain, spasm and tenderness
rmal	"	—	—	—	—	—		Tenderness and swelling cervical spine
sent	"	Clear	Neg.	Normal	0	—	Proved by course of the disease	Hyperesthesia Weakness of legs
reased	"	—	—	—	—	—		Rachitic Rosary Carpopedal spasm
"	"	—	—	—	—	—	Vaccinated 5 days previously	Opisthotonas
—	Left facial	Clear	Neg.	—	0	—	—	—
tive	Left arm " Leg Right face	Bloody	—	—	—	—	Spastic after 11 mo.	
—	Left arm and leg showed paresis	—	—	—	—	—		Choreiform movements Systolic murmur
ctive	Legs	(Showed block)					Incontinent	Loss of sensation in legs Babinski +
J. Absent	Legs Flaccid	—	—	—	—	—	Diphtheria in family	Progressive weakness of legs
. J. Absent	Both legs Flaccid	—	+	Normal	32	—	Operation showed epidural abscess	Anesthesia caudal to umbilicus
—	Facial	—	—	—	—	—	One month later slowly regaining use of muscles	Weakness of legs Dyspnea Pain in back
—	Rt. leg	Increased	+	Reduced	57	—		X-ray showed miliary tbc TBC 1/1000 +
resent	Legs	—	—	—	—	—	Long illness Loss of weight	Cold abscess over thoracic spine
Abnormal	Scattered	—	—	—	—	—	Gradual onset. Biopsy of tumor proved diagnosis.	Ataxic weakness Tumor of rt arm and axilla
—	Pseudo of left leg	—	—	—	—	—	X-ray showed epiphysitis	Soft tissue swelling over left femur
Normal	No	—	—	—	—	—		Cough—Dyspnea Infected throat Substernal retraction
—	Pseudo of left leg	—	—	—	—	—	Operation showed osteomyelitis	Pain and swelling over femur—left
Present	Pseudo of legs	—	—	—	—	—	No orange juice in diet	Pain when handled

the question of accurate technique and clinical and laboratory proof that the disease in question was poliomyelitis must be raised.

In conclusion it may be said that certain diseases simulate poliomyelitis in their signs and symptoms. If, however, the signs and symptoms of poliomyelitis are kept clearly in mind, a careful history and a thorough physical examination of the patient should establish a differential diagnosis. In the few cases where the diagnosis is still in doubt, a lumbar puncture will be a valuable diagnostic aid. If the spinal fluid is negative, poliomyelitis in all probability is not present. In poliomyelitis, as in other syndromes, there is always the possibility of the rare atypical case. The great majority of cases, however, will present a definite clinical syndrome.

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CONGENITAL COMPLETE HEART BLOCK

Report of An Additional Case

BY JOSEPH WITTER GODFREY, M.D.* AND ROBERT STERLING PALMER, M.D.*

THE differential diagnosis of slow pulse in childhood is practically limited to heart block, either congenital or acquired, and cretinism. If the latter can be excluded, diphtheria, congenital syphilis, tumor encroaching on the bundle of His, and congenital malformations of the heart are entertained as possible causes of the block.

The subject of this report, a male child of nine months, was first seen by one of us (J. W. G.) because of failure to gain weight. The birth weight was ten pounds. At nine months the child weighed only sixteen and one-half pounds. Birth was uneventful; there was no cyanosis noted at that time. Development was normal and the history was negative except for the failure to gain weight.

Physical examination showed an undernourished child of nine months, weighing sixteen pounds. The skin was very white. There was slight but definite rachitic deformity of the chest. The most notable finding was a heart rate of 48 which on crying accelerated to 54. There was a very loud systolic murmur widely transmitted over the chest but best heard in the second space to the left of the sternum. There was no thrill. Three months later, in addition to the systolic murmur, a high pitched blowing diastolic murmur was heard in the same region. The apex impulse and left border of dullness was 5 cm. to the left in the fourth interspace, 1.5 cm. beyond the midclavicular line. The blood pressure determination was unsatisfactory but appeared to be 84 systolic and 42 diastolic. Physical examination was otherwise negative. There were no cyanosis and no clubbing of the fingers or toes. Another examination two months later was essentially the same except that there appeared to be a slight systolic thrill palpable where the systolic murmur was loudest, namely in the second interspace about 4 cm. to the left of the sternum. The faint high-pitched blowing diastolic murmer was well heard. It did not seem to be continuous through diastole. The murmurs did not give the typical humming or machinery sound usually associated with patent ductus arteriosus.

Electrocardiogram showed complete heart block and definite right axis deviation (fig. I). A roentgen ray film taken with the target at seven feet showed a bulge in the left upper border of the heart, in the region of the pulmonic conus. In figure II an outline from this film (dotted line) is superimposed upon an outline from a film taken with similar technique of the heart of a child of nine months, weighing 12 pounds, 13 ounces, also a feeding problem but without other abnormalities.

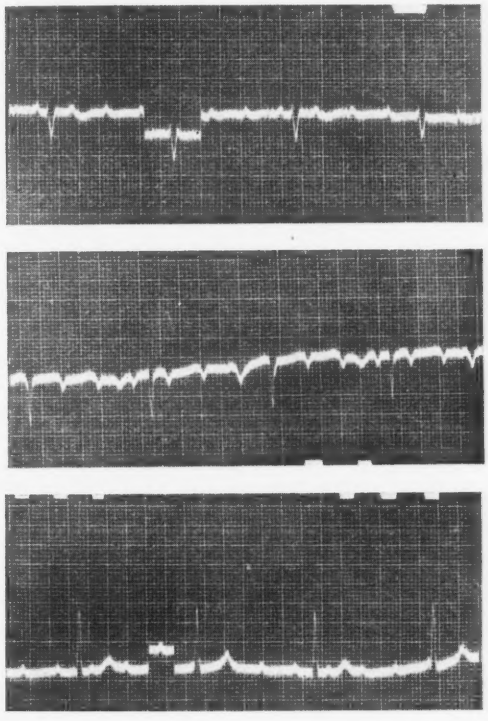

FIG. I. This shows the three leads of the electrocardiogram taken on the case reported. Complete heart block is shown with complete dissociation, the auricles at the rate of 90 and the ventricles at the rate of 50. There is a definite right axis deviation. The standardization in the electrocardiograms is one-half the usual one, that is to say, each five ordinants represent one millivolt rather than ten as is usually the case. This variation was due to the difficulty in obtaining a satisfactory tracing from so young a subject.

The Wassermann reactions on both parents were negative, the child obviously was not myx-

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edematous, and the physical examination clearly revealed a congenital defect in the heart. An accurate diagnosis of the anatomical defect is difficult since the physical signs are confusing. A simple septal defect does not usually cause a diastolic murmur. A patent ductus early in life may cause only a systolic murmur. However, when a diastolic murmur is present as well, the two usually give the familiar and characteristic humming top or machinery murmur. The very slow rate in this case may serve to alter the signs in this respect. A more remote possibility is the presence of an unusual pulmonic defect possibly stenosis accompanied by a deformed or bicuspid

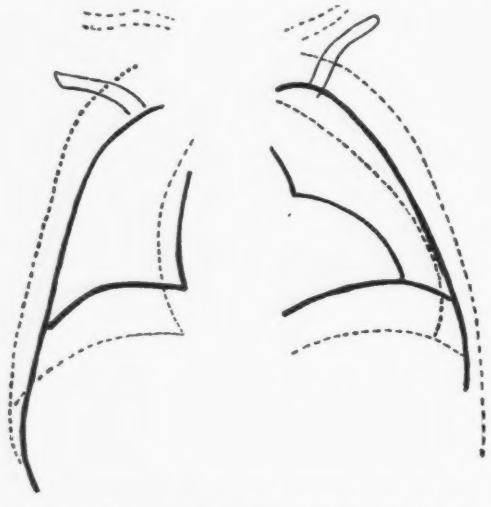

FIG. 11. This shows the outline (dotted line) of the heart shadow and chest wall of the subject of this report superimposed upon the outline (solid line) of the heart and chest wall of a control subject, also a feeding problem but with a normal heart on examination. The control patient weighed twelve pounds and thirteen ounces as compared with the sixteen pounds of the subject. Both films were taken with the target at seven feet. The rounded bulging heart shadow of the case here reported is well shown.

valve. On the whole we are inclined to the diagnosis of patent ductus arteriosus. The only autopsied cases are against this diagnosis. The three referred to by White[1] (Wilson and Grant[2], Yater[3], Abbott[4]) and the other autopsied case in the literature referred to by Yater[3], all showed extensive interventricular septal defects at autopsy.

Lampard[5] collected thirty-one cases of congenital heart block not all proved. Twenty-three showed complete and eight incomplete heart block. A full report of the outcome in these cases is not given. However, one case was said to have died on going in swimming though he had enjoyed similar exercise before. "Others had relatively sudden heart failures and died in a few days or weeks." Some infants were reported who lost their heart block while in others it was noticed until they were in their "teens".

Fleming and Stevenson[6] reported two cases each with probable interventricular septal defects, a male aged three and one-half years and a female aged eight years. The latter may have had the tetralogy of Fallot. She subsequently died but at what age or in what manner was not known. These authors commented upon the infrequency of attacks of dyspnea, cyanosis, and syncope. Davis and Stecher[7] reported one case and stated that only three deaths were recorded and commented that a lack of more serious complaints was surprising. This made the number of cases proved by graphic records twenty. Leech[8] reported a case of complete block in a girl five and one-half years of age, the associated congenital lesions in his opinion being a patent ductus arteriosus though interventricular septal defect was considered. From anatomical considerations, as well as the reported electrocardiograms Leech concluded that the defect producing the block in such cases must be well up in the stem of the bundle. In Yater's case[3] the bundle was completely separated from the node. The heart block may not necessarily be related to the septal defect so often accompanying it except that the bundle may be abnormally subject to infection, or pressure induced by stretching. The latter possibility was proposed by Lampard[5] in view of Smith's case quoted by him which showed no block in forced expiration when, presumably with a more transverse position, there was less tension and pressure on the conducting bundle.

The lack of definite data from the reported cases makes the prognosis difficult to estimate accurately. When Lampard[5] reviewed the literature (1928) there were nine cases over ten years of age, the oldest being twenty-three. The cases reported since then (Davis and Stecher[7], Leech[8], Koenen[9] and Yater[3]) have been under ten years of age. The outlook would seem to be unfavorable from the data available but this is not necessarily so since complete heart block alone may be well borne provided undue demands requiring acceleration of rate are not placed upon the heart. An intraventricular septal defect or a patent ductus arteriosus may likewise be well borne. The combination of either of these and complete heart block naturally does not improve the prognosis of either. Generally speaking, the more severe the associated cardiac anomalies presumably the worse the outlook. Neither a simple intraventricular septal defect nor a patent ductus arteriosus is in itself necessarily serious aside from the hazard of subacute bacterial endocarditis. Tentatively one might limit the life of such children to ordinary activities, excluding competitive games and advise the parents to direct their training to sedentary occupations.

Congenital heart block is rare. Yater's[3] critical review of the literature in 1929 revealed 30 cases, 22 with complete heart block, 6 with partial heart block, alternating between complete and incomplete in one and undetermined in one.

SUMMARY

A case of congenital complete heart block, the twenty-third such case in the literature, is reported.

A probable patent ductus arteriosus is an associated congenital cardiac anomaly. An unusual congenital deformity of the pulmonic valve is a less likely possibility.

There are insufficient data in the literature to make an accurate prognosis, though it certainly is not absolutely bad.

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ADDISON'S DISEASE WITH DIABETES MELLITUS

BY WILLIS M. GOWEN, M.D.*

IN 1885 Thomas Addison published his investigations of the disease of the adrenal glands which now bears his name. His monograph described the condition as an idiopathic anemia related to disturbance of adrenal function, characterized by bronzing of the skin and mucous membranes, with digestive and nervous disturbances leading to cachexia, and terminating fatally. The condition was well described and the views today are essentially the same as those held by Addison, or those introduced later by his contemporaries. From time to time case reports have appeared in the literature and the impression has been that it is a rare disease. It is surprising how often a classical case of this condition escapes recognition, doubtlessly because of the impression of the rarity of the disease. Today it is being recognized more frequently and the consensus is that it is more common than has generally been supposed. This statement is supported by the increased number of cases that are reported in the medical journals.

Without going into details as to etiology, symptoms, diagnosis, and treatment, a brief review may help in understanding the unusual case that recently was treated on the Third Medical Service at the Boston City Hospital, when diabetes mellitus complicated an Addison's disease. The onset is usually insidious, yet acute onset is not rare. The chief complaints are weakness, and this asthenia is one of the dominant features and the earliest symptom arising in the disease and should be carefully investigated when present, muscular fatigue especially. Next to asthenia, pigmentation is the most important finding. It may appear anywhere on the body, but more particularly on the face, neck, backs of hands, and on surfaces most readily irritated. The color is yellow at first, but later various shades of brown appear. Gastrointestinal complaints such as nausea, vomiting and abdominal cramps are prominent symptoms, with emaciation resulting in part from the inability to take and digest food properly. Circulatory weakness is always present and it is not uncommon to find a systolic pressure as low as 50 mm. Hg. Nervous symptoms such as psychic and emotional abnormalities are common and probably numerous individuals have been labelled psychoneurotics, while in reality they are unrecognized cases of Addison's disease. Some regard the blood changes as inconstant, but secondary anemia and diminished blood sugar seem to be the routine finding. The basal metabolic rate and body temperature also are characteristically low. Rapid strides toward treatment for this condition have been made in the past two years. Rowntree, Greene, Swingle, and Pfiffner in the *J. A. M. A.* January 24, 1931, reviewed the forms of treatment from the Muirhead regimen in 1920 to the use of the aqueous extract of the supra-renal cortex announced by Swingle and Pfiffner in March, 1930. It is hoped that the aqueous extract will soon be recognized as a boon as great to those with this condition as insulin was to the diabetic. The following case report presents the typical Addison's disease from many angles, although diabetes mellitus complicated the picture.

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CASE REPORT*

On April 15, 1931, a 54 year old white widow entered the hospital complaining of fatigue.

Family History:

Mother died at age of 70 with "jaundice". No history of tuberculosis or diabetes. Family history otherwise negative.

Past History:

Essentially negative. Married 34 years. Never pregnant. A gradual loss of about 30 pounds in weight during past two years.

*This case was studied on the Third Medical Service, Boston City Hospital. Dr. Cadis Phipps (Physician-in-Chief) has given his permission for the publication of this report.

Present History:

She was perfectly well until February, 1931, at which time her husband died with heart trouble. Following this she developed an "all gone" feeling, and became very weak, easily tired and fatigued on slightest exertion. She no longer had the same desire to do her housework as she previously had, her strength seemed gone, and she became disinclined to any physical effort. This state progressed to utter exhaustion. Her memory and reasoning became blunted. She would have abrupt attacks of sorrow and melancholia, at times crying aloud for hours. At this time friends called her attention to the discoloration of the skin of the face and neck. Several weeks later her appetite became poor and her bowels sluggish, and she became nauseated and vomited. These symptoms were at first mild and intermittent, but gradually cramp-like pains developed over the entire abdomen and vomiting became more constant. At this stage she sought medical advice. An x-ray series of the gastro-intestinal tract was negative, as were gall bladder studies. Finally the nausea, vomiting and abdominal pain recurred so markedly that she entered the hospital.

Physical Examination:

The patient was a well-developed but poorly nourished female lying flat in bed, appearing drowsy and very weak. Skin: The skin was cold and dry. There was a universal brownish discoloration with increase of intensity on the dorsum of the hands and forearms, about the extensor surfaces of the elbows and knees, and about the ankles, face and neck, the color being best likened to that of bronze. Skull and scalp: Normal. Hair: Gray, dry, thin, and easily broken. Eyes: Pallor of conjunctivae, no icterus. Ears: Nose: Negative. Mouth: Lips pale. Teeth absent. Tonsils and pharynx normal. There was a yellowish hue to the soft palate but no definite discoloration or pigmentation of the mucous membranes. Neck: Thyroid palpable. No adenopathy. Thorax: Symmetrical. Marked supraclavicular and intercostal retraction. Heart: Not enlarged to percussion. Sounds distant and weak. No murmurs. Lungs: Other than increase in vocal resonance and tactile fremitus over both apices, lungs were normal. Abdomen: No tenderness or masses. No organs palpable. Genitalia: No pigmentation of mucous membranes. Extremities: Reflexes normal. No edema or cyanosis. Hands and feet very cold. Sensations: Normal. T. 95. P. 80. R. 18. B. P. 84-54.

Brief summary of important evidence: In the history we find that a condition is present that came on insidiously in a female past the middle age of life, characterized by asthenia, lack of interest, digestive disturbances, psychic and emotional abnormalities, pigmentation of the skin and loss of weight. Physical examination revealed marked emaciation; diffuse pigmentation; signs at both apices; dry, cold skin; subnormal temperature, and B. P. 84-54.

Laboratory Data on Entrance:

W. C. 7,000. R. C. 3,500,000. Hg. 55. Urine: S. G. 1010. Yel. Acid. Sug., complete reduction. No acetone or diacetic acid. Sed., negative. Blood sugar, 166 mgms. Basal Metabolic Rate —34. Stools, normal. Sputum, negative for Tb. Sugar tolerance test, typical diabetic curve. X-ray Examination: Chest showed old fibroid tuberculosis of both apices. Flat abdominal plate showed no calcification of the adrenal bodies. Gastro-intestinal series was normal. Gall bladder studies normal. Renal function tests normal. Icteric indices normal. Non-protein nitrogen and cholesterol normal.

Course in Hospital:

This case was considered to be one of Addison's disease with diabetes mellitus. A modified Muirhead regimen was instituted and the diabetes was treated by diet. At the end of two weeks the glycosuria had disappeared and the blood sugar was 97 mgms. The patient was less apathetic and the gastro-intestinal disturbances had subsided. In order to increase the caloric intake we decided to use insulin and within one hour of the first dose of five units she had an insulin reaction. The blood sugar at this time was 70 mgms. Returned to a house diet she again showed complete reduction of the urine and a blood sugar of 200 mgms. Sugar tolerance again showed a diabetic curve. Later several attempts were made to give her insulin and each time she had a tremendous reaction. This idiosyncrasy is characteristic of Addison's disease. Finally we decided to return to the previous diet rather than have repeated insulin reactions. From then on she was sugar free. During this interval she was on thyroid, adrenalin, and whole gland suprarenal extract, and on May 7 the basal metabolic rate come up to —31. She was put on iron and the R. B. C. came up to 5,000,000 with Hg. 70. At this time she was gaining three pounds each week, and on May 25 the basal metabolic rate was —13, so she was allowed up and about the ward, active, happy, contented, and feeling the best she had in four months.

On discharge, July 11, 1931, she was markedly improved, had gained weight, the basal metabolic rate had risen to +1, temperature was normal, diabetes controlled, and she was free from asthenia, gastro-intestinal upsets, and the emotional instabilities with which she entered. She still had the bronzing and pigmentation however.

On August 12, 1931, six weeks after discharge, she again was brought to the hospital in a semi-stuporous state. Her body temperature was low, she was vomiting, her respirations were shallow, and her heart sounds were inaudible. B. P. 80-60. She showed complete reduction of sugar, and acetone. B. S. 262. She was given stimulation and treated for the acidosis, but expired shortly after entry. We learned that after discharge she failed to follow the course of treatment we had advised.

Our final impression of the case was Addison's disease, with diabetes mellitus, and fortunately post-mortem examination was permitted from which studies our clinical impression was confirmed. I am indebted to Dr. M. S. Shiling of the Pathologic Department of the Boston City Hospital for the details of his findings.

SUMMARY OF PATHOLOGIC FINDINGS

Autopsy performed 15 hours postmortem.

Grossly, the only abnormal features noted were a moderate, generalized arteriosclerosis, bilateral healed pleurisy, a light brown diffuse pigmentation of the skin, most marked over the extremities, and a decrease in size of the adrenals. Each adrenal measured about 3.0 cm. in length, 1.0 cm. in width and 0.2 cm. in depth. On section a narrow rim of yellow cortex was made out, with the center of the adrenal a uniform black in color and fairly firm in consistence. No normal milky white medulla was seen. The thyroid was small, firm, grayish brown in color and contained very little colloid. The liver weighed 1000 gm. and aside from a slight amount of scarring just beneath the capsule and along the edge of the liver, the parenchyma was entirely negative.

Microscopically, the important findings were as follows:

Liver: With the routine eosin-methylene blue stain the liver cells everywhere showed a marked rarefaction of the cytoplasm with many of the nuclei vacuolated. With the Best's carmine stain for glycogen, the liver cells were full of closely packed red droplets, and many of the nuclei contained varying numbers of similar droplets. An iron stain was

negative. There was a slight lymphocytic infiltration of the periportal stroma.

Pancreas: Some of the islands of Langerhans were completely hyalinized and, in addition, a few showed a marked lymphocytic infiltration. With the gentian violet stain for amyloid the hyalin material in the islands was positive for amyloid. There was some increase in connective tissue stroma throughout the pancreas.

Kidney: A routine section showed the cytoplasm of the cells lining Henle's loops to be very clear. With the glycogen stain a few of these tubules were seen to contain a moderate number of red droplets in the cytoplasm of the cells lining them.

Adrenals: Sections from both adrenals presented practically the same histologic picture. The normal architecture was completely lacking. The remaining adrenal tissue, which was far less than normal, was collected in a few discrete nodules, in which the cells were arranged in an adenomatous form. The individual cells were greatly enlarged and contained huge nuclei, some of which measured about 25 micra in diameter. There was a great deal of granular greenish black pigment in the cells. Between the nodules were small, scattered clumps of cortical cells in a stroma of dense connective tissue, infiltrated with many lymphocytes. No medullary tissue could be made out in any of the sections.

Thyroid: A few of the lobules consisted of acini, lined with normal cuboidal epithelium and containing a moderate amount of colloid. The majority of the lobules, however, were infiltrated by much lymphoid tissue, which replaced many of the acini and compressed those remaining. The latter were small and practically free of colloid. There was a moderate lymphoid infiltration of the trabeculae.

The remaining organs, including ovary and hypophysis, were not remarkable.

Diagnosis:

1. Sclerosis of the adrenals with compensatory hyperplasia and hypertrophy of the remaining cortical cells.
2. Amyloidosis of some of the islands of Langerhans.
3. Increase in glycogen in the cytoplasm and nuclei of the liver cells with a moderate amount in the cells lining Henle's loops of the kidney.
4. Marked lymphocytic infiltration of thyroid.
5. The findings were consistent with a clinical diagnosis of Addison's disease and diabetes mellitus.

In a brief search of the literature I find that two such authentic cases were reported from the Mayo Clinic's "Staff Meetings of the Mayo Clinic" 5:349-350, December 3, 1930. There are also six other cases reported in the literature where diabetes mellitus has coexisted with Addison's disease.

A RECORD OF PROGRESS*

The Ninth Annual Report of the British Empire Cancer Campaign, which has recently been published, records steady if unspectacular progress. It contains a reasoned account of work which has led some observers to look on cancer as a change in the body cells rather than as an expression of the reaction of these cells to invasion of the body, and it discusses the work now being carried on in this country (England) upon the origin of tumors and upon the inhibition of growth. The impression is left that the body possesses some mechanism of its own whereby resistance is offered to the spread of cancer, and the hope is aroused that it may be possible in the future to stimulate this natural resistance. The curious fact that "mustard gas" prevents the development of cancer in animals which have been treated with cancer-producing substances deserves attention in this respect, for it is believed that the action of the gas is not that of a chemical antidote but of a biological stimulant of some kind. The gas, in other words, appears not to rob the cancer-producing substance of its power, but to give an added power of resistance to the animal's skin. This work will doubtless be subjected to prolonged examination of the most critical kind before final conclusions are reached.

Meanwhile there is no doubt that the workers at the Cancer Hospital have obtained a synthetic product possessed of a very high degree of cancer-producing power. This product, called Dibenzanthracine, gives origin to tumors in a fashion which is awe-inspiring, and makes it certain that the body's resistance to tumor formation can be overcome by outside agencies possessed of certain qualities the nature of which remains obscure. The body's resistance, nevertheless, can be so far fortified by "mustard gas" that even Dibenzanthracine fails to produce cancer. The way has been opened therefore to a study of resistance and of the elements, whether chemical or biological or both, on which it depends. Attention naturally turns from this work to the studies in the inhibition of growth now being carried out at the London Hospital. The claim is made that a high percentage of spontaneous tumors in mice were caused to disappear by the use of a substance (Euglobulin) possessed of cancer-resisting qualities. The temptation to relate the two lines of research is obvious, but there is no justification at present for yielding to it. Indeed it must be urged that so far all this work is in a preliminary stage. Application to human beings is out of the question until much further research has been accomplished.

*Extract from the *London Times*, July 13, 1932.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending September 10, indicate a mortality rate of 9.0 as against a rate of 10.3 for the corresponding week of last year. The highest rate (17.8) appears for Miami, Fla., and the lowest (4.1) for Lynn, Mass. The highest infant mortality rate (22.4) appears for Miami, Fla., and the lowest for Camden, N. J., Canton, O., Des Moines, Iowa, Duluth, Minn., Erie, Pa., Evansville, Ind., Knoxville, Tenn., New Bedford, Mass., New Haven, Conn., Syracuse, N. Y., Tampa, Fla., and Waterbury, Conn., which reported no infant mortality.

The annual rate for 85 cities is 11.3 for the thirty-six weeks of 1932, as against a rate of 12.2 for the corresponding period of the previous year.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18391

A CASE OF SUDDEN DEATH. WAS IT HEART DISEASE?

MEDICAL DEPARTMENT

A sixty-eight year old retired granite worker went to a private physician complaining of shortness of breath and swelling of the abdomen.

He had been well until the onset of symptoms, a month before death, and did not seem at all seriously ill. He had never had edema of the ankles at any time. He had not been exposed to silica for some years.

His past history was negative except for several attacks of painful swollen joints during the past twenty years. He had never been a heavy drinker. There was no history of lues. The abdominal swelling of which he complained was chiefly in the umbilical region.

Examination by the physician showed slight generalized adenopathy, most marked in the inguinal nodes. There was an itching rash with crusted lesions, especially in the inguinal regions and over the abdomen, apparently scabetic. The heart was not enlarged. There were no murmurs. The blood pressure was 160/90. The breath sounds were very noisy. The abdomen was enlarged in the umbilical region. There was no evidence of fluid. The liver was enlarged, soft and not tender.

The temperature was 99° to 100.5°, the pulse 120. The respirations were 30 to 40.

The patient remained up and about the house until the last two weeks, which he spent in bed on account of increased shortness of breath. The day before death, because of signs of ascites as well as the increasing dyspnea, an internist was called in consultation. Plans were made to send the patient into the Massachusetts General Hospital for further study. The following morning he was found dead in bed.

CLINICAL DISCUSSION

DR. MAURICE FREMONT-SMITH: I should like to know all sorts of things about this patient. Did he have leukemia? The spleen is not noted as being enlarged, yet I wonder whether that might be a possibility. He may have had lymphoblastoma. I have seen it in a man of sixty-five whom Dr. Mallory saw with me, and of course it is well known that Hodgkin's does occur in older people.

This swelling in the umbilical region means nothing to me. I wonder if he ever had ascites.

It really comes down to a man of sixty-eight who complains of shortness of breath and swelling of the abdomen and then suddenly dies. I think any diagnosis is a pure guess. He probably died of a cardiac accident. The chances are that he had some small cardiac infarcts and then later a larger one. That seems to me a good possibility. Of course it does not explain his adenopathy. I should be inclined to make that diagnosis in addition to any other, such as leukemia or Hodgkin's.

DR. RUSSELL F. SHELDON: You asked about that swelling of the abdomen. The only swelling was that, as one looked at it, it was round. The liver edge was felt about two inches below the costal border.

DR. FREMONT SMITH: Was there any other swelling?

DR. SHELDON: Just around the abdomen. The umbilicus was the high point.

DR. FREMONT SMITH: In other words a little increase in fat with weakness of the recti muscles might give that picture.

DR. SHELDON: The liver was not very hard or soft or nodular. It was merely a resistant mass with a definite edge.

DR. FREMONT-SMITH: You could not feel the spleen?

DR. SHELDON: I could not feel the spleen.

DR. FREMONT-SMITH: What was your diagnosis?

DR. SHELDON: My first thought, as you said, was that he was a cardiac. That was the one thing which might explain everything. There were the chest and abdominal conditions. An internist came in and said, "No, he isn't a cardiac at all; the heart is perfectly normal. These chest signs might mean an inflammatory condition. I do not know what he has. Send him to the hospital and find out." The next morning he died.

DR. MAURICE FREMONT-SMITH'S DIAGNOSIS

Possibly leukemia or Hodgkin's disease.
Probably cardiac infarct.

ANATOMIC DIAGNOSIS

Lymphoblastoma, Hodgkin's type.
Fatty infiltration of the liver.
Atheroma of the aorta, moderate.
Anthracosis of the lungs.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: We found a potentially lethal disease that he had, but we did not find much to suggest that he actually died of it. He had Hodgkin's disease with a very slight generalized glandular involvement but a fairly marked enlargement of the retroperitoneal and mesenteric glands which I think accounts for the local prominence. The spleen was barely above

normal size. The liver was distinctly enlarged, but he had no Hodgkin's in it, only a certain amount of fatty infiltration.

His heart weighed 350 grams. The coronary arteries were perfectly smooth, entirely negative. There was only a very moderate degree of atheroma in the aorta.

The lungs showed a very marked degree of anthracosis. Most recently he had been a janitor. Before that what was his occupation?

DR. SHELDON: He had been a moulder and worked in sand.

DR. MALLORY: It was probably both anthracosis and silicosis in equal degrees. There was a slight patch of fibrinous pleuritis in the left lower lobe. There was a mere trace of bronchopneumonia.

DR. FREMONT-SMITH: Do you feel that a person could die of coronary spasm with no positive findings in the coronaries?

DR. MALLORY: I am doubtful of it. I have seen a great many cases where there had been death with coronary symptoms in which we could not demonstrate complete closure of the coronary artery, that is, no thrombosis, but invariably we found well marked arteriosclerosis with at least moderate narrowing of the artery, not a perfectly smooth one such as we found in this case.

We are left, as we often are, with no explanation of the sudden death. I should say that forty per cent of sudden deaths we can not explain.

CASE 18392

MALNUTRITION, VOMITING AND INFECTION IN AN INFANT

CHILDREN'S MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. RALPH W. DAFFINEE:* A white female infant five weeks old entered the hospital on December 26. The chief complaint was vomiting.

The father, mother and one brother five years old were well at the time of the patient's entry to the hospital.

Delivery had been instrumental and at term in a local hospital. The birth weight was 6 pounds 10 ounces. The infant seemed normal in all respects. During the last two weeks of pregnancy the mother suffered from toxemia and had a high blood pressure. The infant was breast fed for four days and seemed normal. Then she began to vomit ten to thirty minutes after each feeding. The vomiting was not projectile. At times the whole feeding was regurgitated, at other times only a portion. Milk was expressed from the mother's breasts and fed to the child from a bottle, but the vomiting continued.

On discharge from the maternity hospital the infant was fed regularly on a formula of cow's milk 12 ounces, water 5 ounces and Karo syrup 1 ounce, 2 ounces every three hours, as the mother's milk had failed. The child continued to vomit.

The bowels were normal until one week before admission, when the infant became constipated and was given a dose of castor oil. On three occasions during the week the mother noticed thin streaks of blood in the stool. The morning of admission the baby vomited dark brownish fluid which resembled argyrol.

No fever had been noticed at any time. The baby had coughed a little during the past few days. On admission to the hospital she weighed 5 pounds 14 ounces.

Physical examination showed a poorly nourished, restless infant, fretful and not acutely ill. The respirations were normal and the color was good. The infant seemed hungry. The fontanels were open and not bulging. The head was negative for infection. The neck showed no stiffness. The lungs were clear and resonant. The heart was negative. The abdomen was soft throughout, not distended and showed no tenderness. The spleen was not palpable. No tumor or masses were felt. There were no peristaltic waves at the time of the entrance examination, but later the infant was observed while feeding and definite peristaltic waves were seen. The extremities were negative. The labia majora seemed a little edematous but not inflamed. There was no vaginal discharge.

X-ray with barium showed rapid passage of part of the meal into the duodenum. This passed normally into the intestines. Four hours later there was still considerable residue in the stomach.

For the first four days the child's temperature was elevated on occasions to 101° and once to 104°. On December 30 it became normal and continued to be so until January 5. Then for four days fever was occasionally present (101° to 102.8°). Then normal temperature returned until January 13. From then until January 24 the temperature rose as high as 101° each day.

The plan of treatment for the vomiting at first outlined was:

1. Gastric lavage every four hours, followed by gavage of breast milk.
2. Refeeding if food was vomited.
3. Maintenance of body fluids by clyses of normal saline.
4. Atropine again 1/500 at time of feeding.

This was continued until January 3. Two clyses were given during this time. Only 12 ounces of breast milk was given daily (about 40 calories per pound). The child vomited about one-third of the feedings. The vomiting was found to be projectile. On January 3 the weight suddenly fell to 5 pounds 9 ounces. Then breast milk was concentrated from 18 ounces to 12 ounces by boiling and 2 ounces given every four

*Recently resident on the Children's Medical service.

hours. The child maintained her weight until January 8, but did not gain. She continued to have some vomiting.

Then followed thick feedings. The following prescription was ordered: farina 1 ounce, breast milk 9 ounces, dextrimaltose 3 ounces, cooked until thick; six feedings at intervals of four hours. Three hours after each feeding one ounce of water was given. She took and retained the thick feeding well, but vomited the water. Her weight remained stationary, being 5 pounds 10 ounces on January 15. At this time she began to refuse the feedings. She had an infection and a few râles in her chest. She was again put on concentrated breast milk feeding and atropine, but vomited it. On January 19 concentrated feedings were again instituted and a clysis given. In three days the patient's weight had increased to 6 pounds 7 ounces although her general condition was unchanged.

On January 21 the right ear drum was seen to be red and bulging. A paracentesis was performed. Blood and pus were obtained. On January 22 an area of skin the size of a quarter dollar in front of the right ear was seen to be red, with a raised edge. The border spread over the cheek the next day. The center tended to fade. The temperature rose to 103°. On January 24 the erythema had spread to the neck. The temperature had then fallen to normal. The child died the next morning.

Clinical Discussion

Dr. Harold L. Higgins: This case points very much toward some obstruction of the pylorus. One is inclined to feel that perhaps we are dealing with a pyloric stenosis. The patient is a girl. Pyloric stenosis is more unusual in girls than in boys, but it does occur in girls. There was considerable trouble with the feeding of the child from birth. She seemed to vomit practically every feeding, although the vomiting was not regarded as projectile. She was fed from a bottle instead of at the breast and the vomiting persisted. When she was changed to a formula the vomiting persisted, and continued with every type of feeding given to her while she was in the hospital. That is quite in keeping with pyloric stenosis or pylorospasm. It is pretty obvious that she was getting some food through her pylorus, because her stools apparently were normal in size and number. She had been able to maintain her weight, although she had never recovered her birth weight. She was not continually going downhill and was not presenting distinctly a problem of inanition. There was some opening of the pylorus as seen by the x-ray. Some of the barium was seen to pass through immediately. No tumor was felt. One expects a tumor in pyloric stenosis, but often none is felt. The problem might arise as to whether there was pyloric stenosis or pylorospasm. If there was pylorospasm one would expect that some of the meals would go through satisfactorily and that some would not. It is quite true that pyloric stenosis and pylorospasm frequently go together.

After she had been in the hospital a few days a diagnosis of probable pyloric stenosis was made and apparently the decision was reached to treat it medically. Medical treatment seemed justified first because the child had a slight infection and secondly because some food was passing through the pylorus. So atropine was given to overcome any possible tendency to spasm. Atropine has been used with some success, although it is not a specific by any means. She had lavage and gavage of breast milk, and she vomited. On some occasions when she vomited a good deal and seemed to become dehydrated, she was given clyses. With this treatment she was getting only 25 to 30 calories per pound per day. We can tell that things were not going well because she was losing weight.

Some other method of treatment seemed indicated, so concentrated breast milk was tried. Eighteen ounces was concentrated to twelve; that is, each ounce of breast milk contained 30 calories instead of 20. Less fluid was given, therefore the child was not likely to vomit so much. That was successful to the extent that she held her weight; but she did not gain.

Then thick feedings were tried. She was given a formula which was recommended by Sauer, farina, breast milk and dextrimaltose cooked into a thick cereal. It seems to me that the caloric value of the feeding was pretty high, 120 calories in the farina, 180 in the breast milk and 360 in the dextrimaltose, making a total of 660 calories, or 110 calories per pound of body weight. Perhaps the entry of 3 ounces of dextrimaltose is incorrect; it may have been drams. When taking solid food the child must also take fluid. Therefore water was given three hours after the feedings in the hope of getting in sufficient fluid in that way.

The child's weight was constant until January 15, when she began to fail to eat. Then they changed the feeding back to concentrated breast milk and atropine, but she did not retain that. Throughout this whole procedure the child had had intermittent fever. Unfortunately she was born in December. December, January and February are the months of the year when there are many respiratory infections, and apparently this child was never quite free from them. She would go for about a week without them, then run a slight fever for a while, apparently in the nature of a minor respiratory infection.

Eventually she developed a red ear. A paracentesis was done for that and she developed erysipelas apparently. She did not have the resistance to fight it off. Her temperature fell to normal and she died.

My diagnosis in this case is probable pyloric

stenosis of a mild degree with some pylorospasm. There is right otitis media, erysipelas and malnutrition.

This is the unfavorable picture of what happens sometimes in cases of pyloric stenosis that one tries to treat medically. With the child who is treated medically we have considerable difficulty in getting a satisfactory gain in weight, and being undernourished the child is especially prone to infections and to have poor resistance to them.

The question as to whether the child should have been operated on while under our care is somewhat open to doubt. She was certainly running an infection and I think good clinical judgment was shown in not operating in haste on a child who had an infection and in waiting to see if things would not clear up with the clearing up of the respiratory infection.

Here are the x-rays of the stomach. Some of the barium has passed through the pylorus in the first one. You see also there still is some food in the stomach at the end of three hours.

CLINICAL DIAGNOSES (FROM HOSPITAL REPORT)

Malnutrition.
Pylorospasm.
Nasopharyngitis.

DR. HAROLD L. HIGGINS' DIAGNOSES

Otitis media.
Erysipelas.
Pyloric stenosis.

ANATOMIC DIAGNOSES

Malnutrition.
General peritonitis, streptococcus hemolyticus.
Bronchopneumonia, streptococcus hemolyticus.
Empyema, left pleural cavity.
Hemosiderosis of the spleen.
Acute enteritis.

Pathologic Discussion

Dr. John I. Bradley: The autopsy findings are as follows. The body was well developed but emaciated. There was swelling and redness over the right cheek and jaw. The peritoneal cavity contained 300 cubic centimeters of thin watery light yellowish fluid containing small masses of fibrin. There was a thin film of fibrin over the retroperitoneal tissues and there were numerous fibrinous adhesions between the coils of intestine. The left pleural cavity contained approximately 600 cubic centimeters of thin yellowish fluid containing masses of fibrin. The left lung was collapsed to approximately one-quarter its normal size. The pleura was coated with a thin sheet of fibrin. The lung was completely atelectatic. No area of consolidation was found. On examination of the stomach the pylorus was not constricted and the muscle apparently not thickened.

Microscopic examination showed an exudate of polymorphonuclears in the bronchi and in a few of the adjacent alveoli,—early bronchitis and bronchopneumonia. Section of the intestine showed polymorphonuclears in the lymphatics, the mucosa and the muscularis. Gross diagnoses of general peritonitis, acute fibrinous pleuritis, bronchopneumonia and acute enteritis were made. Cultures from the pleural cavity, the lung and the peritoneal cavity yielded hemolytic streptococcus.

Dr. Higgins: On what do you base your diagnosis of acute enteritis?

Dr. Bradley: On the microscopic findings—the polymorphonuclear infiltration.

Dr. Higgins: Wouldn't that be peritonitis rather than enteritis?

Dr. Bradley: It was in the mucosa and in the muscularis, and I think extension in that far is more than peritonitis would account for. The lesion, though, was not evident in gross and was not an outstanding picture. From the pathologic evidence we see that the pyloric condition was one of spasm.

Dr. Higgins: From your record can you make out whether any peritoneal infusions were done?

Dr. Bradley: There is no record of any puncture marks over the abdominal wall. I think one might reasonably postulate a generalized infection and peritonitis from the generalized infection above.

Dr. Higgins: The child had been having a cold. She got an otitis media which presumably might be streptococcus, and developed erysipelas from it, and from this she got a streptococcus septicemia; and with streptococcus in the blood we would be likely to find pus forming in various places. If she had been having intraperitoneal infusions and still had some fluid left in the peritoneal cavity it is not to be wondered at that it became purulent. Apparently an effusion like this can come on quite quickly. A streptococcus empyema may develop almost overnight in a child if it has bronchopneumonia. I do not think that this pleuritis is necessarily of long duration, or even the peritonitis. It is quite obvious from the record that both the pleuritis and the peritonitis were missed clinically; the symptoms seemed to be covered by the diagnosis of erysipelas and malnutrition. It is quite noticeable that she gained a good deal of weight a few days before death; possibly she developed at that time the effusion which would explain the increase in weight.

A Physician: If the peritonitis was of recent origin, what caused the vomiting almost a month before death?

Dr. Bradley: I think that was due to pylorospasm.

A Physician: There were streaks of blood in the stool in the beginning, too.

DR. HIGGINS: Apparently that was not found when she came to the hospital. Of course streaks of blood in the stool would have to come from somewhere. They may come from a fissure around the anus; they may come from ulceration of the bowel in some place. If that had persisted, especially if the stools had contained much mucus, one would have suspected dysentery.

DR. BRADLEY: We found no gross lesion such as one might expect with gross blood in the stools.

A PHYSICIAN: Couldn't the conditions be explained on the basis of infection? It may have been infection from the very first and the child may have appeared to hold her own until repeated reinfections lowered her resistance.

DR. HIGGINS: Possibly she had almost from birth a streptococcus infection which interfered with the handling of food, every once in a while led to a temperature, and eventually blossomed forth as erysipelas and septicemia?

DR. BRADLEY: It is really impossible to estimate the total duration of the streptococcal infection. Certainly the major manifestations that we found were recent, but as to the total duration I am sure I cannot say.

DR. HIGGINS: How old do you think the peritoneal and pleural effusions were?

DR. BRADLEY: I should estimate forty-eight hours.

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THE ORIGIN OF SYPHILIS

FEW diseases have aroused as much interest and speculation regarding their origin and epidemic spread as has syphilis. From the purely epidemiological standpoint there is strong evidence for the transportation of the disease from the New World by Columbus' sailors. The high virulence of the disease when first noted in Europe and its almost explosive spread strongly suggest that the organism was attacking a virgin field, devoid of protection through previous contact with the parasite. Opposed to this view have been the findings of supposed syphilitic bones from ancient graves in Europe and Egypt. Williams[1] has presented a most valuable contribution to this subject in the form of an elaborate review, liberally illustrated, of all the important bones supposedly showing evidence of syphilis recovered in the course of archeological investigations. The conclusions which he draws are of great importance and should go a long way toward settling this still debated question. He finds that all of the trans-Atlantic discoveries of bone, supposedly syphilitic, are open to question, either as to diagnosis or as to true antiquity of origin. He stresses the difficulty of differentiating in some of these specimens a true syphilitic process from osteomyelitis and certain of the osteodystrophies.

In contrast to the trans-Atlantic material numerous finds in diverse parts of this continent, determined with as much accuracy as possible to be pre-Columbian, show unmistakable evidence of syphilis, although, as Williams points out, there is much difficulty in eliminating the possibility of yaws. All in all this review by Williams offers about as conclusive proof as can be obtained for the origin of syphilis in the New World and its transmission to Europe as a result of the Columbian expeditions.

REFERENCE

1 Williams, H. U.: The origin and antiquity of syphilis: the evidence from diseased bones. Arch. Path. 13:779 (May) 1932.

A BLIND SPOT

DUMAS, in the Three Musketeers, quaintly entitles one chapter of his description of the thrilling exploits of D'Artagnan with the phrase "In the dark all cats are gray", and it is good modern psychology to say that we see not things but differences. The artist uses contrast to heighten his effects and if what we think of as white snow, he depicts as really blue or purple, we finally acknowledge that he is right, except possibly as to emphasis. We recognize our physiological blind spots: the blind spots of our mind we refuse to recognize until some striking contrast shocks us into awareness.

The law of Massachusetts with reference to the licensing of physicians has a blind spot in the field of medical education, so that it is scarcely able to discriminate among medical schools except as to mere existence. In the darkness of its night all medical schools look alike to it. Perhaps this is due to inherited tradition. Around the trinity of the church, the town hall and the school the early communities developed. It may be we have forgotten that "the history of institutions is often a history of deception and illusions; for their virtue depends on the ideas that produce and on the spirit that preserves them, and the form may remain unaltered when the substance has passed away."

Outside of Massachusetts, scales have fallen from the eyes, and differences among medical schools have been detected. It has been found that according to educational standards, in the formulation and upholding of which some individuals from Massachusetts have played an important part, certain medical schools are unworthy of the honor of being denominated educational institutions. Outside of Massachusetts, there are none of these United States that do not in their laws express this awareness that there are serious differences among medical

schools and that they will give approval only to such schools as meet certain standards of educational fitness.

That other states take a certain course of action has never been a reason why Massachusetts should take that course; nor has the action of another state ever been regarded as a cogent argument against such action by Massachusetts. The example of other states, and in contrast Massachusetts stands alone, should make us scrutinize carefully our position, to see if we may not have overlooked some important consideration, some significant fact of which others have become aware.

The fact of which the law in other states takes cognizance and of which the law in Massachusetts does not, is that there are medical schools which confer the degree of doctor of medicine upon candidates who do not deserve it, taking into consideration generally accepted reasonable standards of what it means to earn the degree. The candidates fulfill the conditions laid down by the school, live through a certain period of time during which they are enrolled by the school, perhaps are given credit for work they never did, take and pass certain perfunctory examinations, and pay certain fees. They are then given a degree which once denoted membership in a learned profession. By what token are these schools to be reckoned worthy of approval?

It is to be hoped that the eyes of Massachusetts also will be opened so that in its law it will take cognizance of this notorious fact, the existence of which is formally recognized in every other state in the Union.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

WESSELHOEFT, CONRAD. M.D. Harvard University Medical School 1911. Associate in Communicable Diseases, Harvard Medical School. Associate Professor of Theory and Practice, Boston University School of Medicine. Physician, Haynes Memorial Hospital and Associate in Research, Evans Memorial, Massachusetts Memorial Hospitals. Address: 315 Marlborough Street, Boston. Associated with him is

SMITH, EDWARD C. A.B., M.D. Harvard University Medical School 1922. Instructor in Communicable Diseases, Harvard Medical School. Assistant Professor of Pediatrics, Boston University School of Medicine. Resident Physician, Haynes Memorial Hospital. Address: Harvard Medical School, Boston. Their subject is "Results of the Use of the Drinker Respirator in Thirty Cases of Respiratory Failure in Poliomyelitis." Page 559.

THAYER, WILLIAM S. A.B., LL.D., Sc.D., Dr. (Hon.) University of Paris. F.R.C.P. (Hon.) Edinburgh and Ireland. M.D. Harvard University Medical School 1889. Professor Emeritus of Medicine, Johns Hopkins University. His subject is "The Chief." Page 563. Address: 1208 Eutaw Place, Baltimore, Maryland.

SHERWOOD, DAVID W. B.S., M.A., M.D. Harvard University Medical School 1927. Member of the Out Patient Staff, Children's Hospital and of the Pediatric Staff, Newton Hospital. His subject is "A Series of Cases Presenting Syndromes Suggesting Poliomyelitis." Page 571. Address: 66 Commonwealth Avenue, Boston.

GODFREY, JOSEPH W. M.D. College of Physicians and Surgeons, Boston, 1890. On Obstetric Service, Union Hospital, Lynn. Associated with him is

PALMER, ROBERT STERLING. A.B., M.D. Harvard University Medical School 1925. Assistant in Medicine, Massachusetts General Hospital and Harvard Medical School. Assistant Cardiologist, Boston Lying-in Hospital. Address: 264 Beacon Street, Boston. Their subject is "Congenital Complete Heart Block. Report of an Additional Case." Page 575.

GOWEN, WILLIS M. M.D. Tufts College Medical School 1930. House Physician, Third Medical Service, Boston City Hospital 1930-1932. Resident Physician, Quincy City Hospital 1932-1933. His subject is "Addison's Disease with Diabetes Mellitus." Page 577. Address: Quincy City Hospital, Quincy, Mass.

The Massachusetts Medical Society

STATED MEETING OF THE COUNCIL

A STATED meeting of the Council will be held in John Ware Hall, Boston Medical Library, on Wednesday, October 5, 1932, at 12 o'clock, noon.

Business:

1. Reading record of last meeting in abstract.
2. Report of Committee of Arrangements for Annual Meeting.
3. Reports of Committee on Membership and Finance.
4. Reports of committees to consider petitions for restoration to the privileges of fellowship. Appointment of new committees for the same purpose.
5. Appointments of Auditing Committee, to audit Treasurer's accounts as of December 31, 1932, and to fill vacancies.
6. Report of Committee on State and National Legislation.
7. Report of Committee on Postgraduate Medical Instruction.

8. Report of Delegation to House of Delegates, American Medical Association.
9. Letter from Secretary and General Manager, American Medical Association, on dual representation in the House of Delegates, A. M. A. of Non-Resident Fellows of the Massachusetts Medical Society.
10. Incidental Business.

WALTER L. BURRAGE, *Secretary.*

Brookline, September 28, 1932.

Councilors are reminded to sign one of the attendance books previous to the meeting.

THE PHYSICIAN, THE LAITY AND CHIROPRACTIC

THE COMMITTEE ON STATE AND NATIONAL LEGISLATION OF THE MASSACHUSETTS MEDICAL SOCIETY

8 The Fenway, Boston.

Dear Doctor:

How can an intelligent voter declare himself properly if he is not familiar with the principles underlying the subject?

Have you been active in asking your patients, nurses, pharmacists and others to vote against the initiative petition of the Chiropractors?

Do you know that one of our members, without disclosing his identity, obtained a degree of "Doctor of Chiropractic" on the payment of one hundred dollars, and passing the examination after eighteen hours of study?

Isn't it a part of your moral code to protect the sick, the injured and the infirm from those unable to comply with the present law?

Are we guilty of inertia?

Very truly yours,

H. G. STETSON, *Chairman.*
T. J. O'BRIEN.
F. E. JONES.
A. W. MARSH.
SHIELDS WARREN, *Secretary-Treasurer.*

MISCELLANY

PRENATAL AND MATERNITY SERVICE*

BY JOHN ROCK, M.D.

I offer my remarks to all who can concern themselves first, in the interest of those wives who expect to become mothers, and secondly, in the interest of public health in which the comfortable and harmless delivery of normal children is a large factor. For a woman to give birth to a child may be considered a natural process, but this does not mean that nature, unaided, will necessarily protect both mother and child from all harm.

Knowledge of how babies are nourished in the womb and of the effect which the baby in the womb has on the mother while she is carrying it, has increased extensively since even our own mother's day. We now know, too, more about the sicknesses to which pregnant women are particularly susceptible; and, what is more important, how many of them may be prevented, or made less severe. We know more about the special dangers to mother and baby during the actual process of delivery, and, in most cases, how to anticipate and avoid these dangers. Lastly, we know a great deal more of whence come the complications following birth, and how they, too, may be prevented.

It is obviously the duty of each husband to provide his wife with all the safeguards which have come from the more exact knowledge of maternity problems. It is the duty of each expectant mother to take advantage of such protection. It is the duty of doctors who accept responsibility for confinement cases to supply their patients with all known safeguards. If they are not equipped with ability, time, and adequate hospital and laboratory facilities to do the work well, they should direct their patients elsewhere so that these women may not ignorantly suffer from the lack of it. This is not only professional ethics; it is simple honesty. It is refusal to "shortweight a customer". For couples who cannot afford to pay the whole expense of such service, semi-charitable agencies must be used, and for those who can afford no part of the expense, the public, for its own economic and social safety, must supply the necessary funds.

Organized society, comprising the inhabitants of any given area, be it town, city, country, or continent, for the safety and efficiency of the group, protects the individual members of the group from as many hazards as possible. Expensive surveys are made of the oceans, rivers, and lands, that men may travel and convey their goods more safely. Expensive weather bureaus are maintained to forewarn individuals at sea, and in the air. Departments of agriculture spend huge parts of the citizens' money to study and spread information about insect and animal pests in order that food and clothing may be produced rapidly and cheaply for all. Numerous men are employed to police every community to protect all individuals from the harmful deeds of the wicked and the ignorant. Along with these other activities, all performed for the protection of life and happiness of the individual, organized society, through city, state, or federal government, studies all cases of health and disease, and does all it can to instruct individuals and protect them from sicknesses and death.

So the government concerns itself intimately with the welfare of all women during pregnancy and confinement. The State of Massachusetts in coöperation with other states has studied carefully the dangers to mother and child, and actively tries to protect the expectant mother from these dangers. During the summer of 1930 the White House Conference of representatives from all the states studied carefully the difficulties and dangers of

*Read over station WBZ, June 7, 1932, for the Massachusetts Committee for the White House Conference on Child Health and Protection.

childbirth, and worked out a plan for the adequate care of the pregnant mother which would anticipate and prevent so far as possible the dangers to which she might be exposed. This plan is called "The Minimal Requirements for Prenatal Care."

A moment ago I called it the duty of each husband to provide his pregnant wife with every safeguard, and the duty of every expectant mother to take advantage of such protection. "The Minimal Requirements for Prenatal Care" will show them the way. The State Board of Health is anxious to supply them with all this information. As residents in the Commonwealth, they are entitled to this service which they pay for in taxes, either directly or indirectly, whether they use the service or not. Anyone, rich or poor, who cares to take advantage of this very helpful service from the state, needs merely to write or telephone directly to the Board of Health, in the State House in Boston, and ask for Maternity Service, or service to women expecting babies. This is very simple and exceedingly worth while.

Now exactly what is the service which the citizens through their "common wealth" offer each other? As soon as the State Department of Public Health learns that a certain expectant mother or her husband, for her, desires to take advantage of the maternity service offered, a private letter addressed to the mother is sent, with a booklet called "Baby and You." This booklet, the product of well-trained and experienced specialists in midwifery, discusses, simply but comprehensively, those matters which concern, so constantly, the pregnant woman and her husband. Paragraphs are written after such headings as these: "Signs of Pregnancy", "General Rules for Keeping Well", "Clothing", "Bathing", "Care of the Breasts", "Diet in Pregnancy", "Teeth in Pregnancy." The second chapter discusses the "Conditions which may Occur During Pregnancy", such as: "Nausea and Vomiting", "Complications", "Bleeding", "Kidney Trouble", and "Overweight." Following are chapters on "Mental Hygiene", "Preparation for Confinement", "Signs of Labor." The latter half of the booklet then discusses matters relating to care of the new baby.

When the first letter with the booklet is sent to the wife, one also goes to the husband. With sympathy and understanding, his attention is called to his triple responsibility—to his wife, society, and himself—during this important period in his life; and his active coöperation in the proper care and protection of his wife is called for. Each month thereafter, his wife receives an encouraging and informative note which speaks of those matters most likely to concern her at the particular time the letter is received.

You will see that all the service is merely educational. An attempt is made to instruct both husband and wife in all the matters which pertain to pregnancy and confinement. Emphasis is laid on those details of daily life and practice which tend to keep the woman healthy and happy, and which, through her, give the growing child in her womb proper nourishment and protection. Her attention is called to the more common disturbances of health and happiness which may arise during pregnancy to jeopardize her own or her baby's welfare; and she is taught how to avoid these disturbances.

Particular effort is made in all these letters and in the booklet to teach the husband and wife what constitutes good medical care, — care which only a doctor can give. Satisfactorily to protect the mother and baby, applying all the current knowledge about maternity problems this medical service should meet "The Minimal Requirements for Prenatal Care" which I mentioned above, the specifications agreed upon by the White House Conference. The first instruction given to the woman and her husband is to visit the doctor who will deliver her, if services can be paid for; or to visit the charity hospital where she can be delivered if money is scarce. The letter and the booklet teach them what care the doctor or hospital should give her, that at all times they may know whether she is receiving at least "The Minimal Requirements for Prenatal Care."

The city and state inspect dairies and examine milk, so that when a housewife buys a quart of milk she may be sure of getting her money's worth of a healthful product. The size of a loaf of bread is determined by law, that, again, the one who purchases may not be deceived. It is not possible for the state to superintend the doctors' work to this extent. It tries to license for practice only those who are capable of giving adequate service, but it has not the means of compelling them to do so. Some are not interested in maternity work, some are too busy to do it well, some cannot afford to take the time from other better paying or more pleasant work, and some perhaps have not kept abreast of progress, and so do not know what "The Minimal Requirements for Prenatal Care" are. To none of these should women entrust themselves for care during pregnancy and confinement. To help them then, the State Department of Health, as agent of the citizens themselves, teaches the willing husband and wife what good care is, so that failing to obtain it from one doctor, they will know it, and put themselves in the care of another before it is too late. This is not State Medicine; it is legitimate welfare work.

Let everyone try to improve maternity work by appreciating the value of this public service, and spreading information about it, and when the occasion arises, let each couple take advantage of it. It is merely necessary to notify the Department of Health in the State House in Boston, that maternity service is desired.

BRINGS POLLEN-FREE AIR TO HOMES

New Low Priced Glass-Wool Air Filter Provides Local Relief for Hay Fever Sufferers

Unusually widespread suffering from hay fever during the current autumn has brought forth an announcement of help from an unexpected quarter. While a million or more victims of this seasonal malady have been seeking relief through medicaments, or escape to pollen-free regions, the industrial field in coöperation with the medical fraternity, has attacked the problem from a preventive angle and come forth with sensational recommendations.

A certain glass company has perfected a glass-wool air filter which can be attached to warm-air furnaces, window or room ventilators, or air conditioning units, and is helpful in providing clean air, free of dirt and soot to homes and buildings.

Experimental tests seem to prove beyond a doubt that these filters also perform the additional function of removing 99 per cent of hay fever pollen from the air. This means that any hay fever sufferer, by a very slight outlay for installation of this equipment in his home, can escape most of the discomfort heretofore looked upon as inevitable in the fall and spring.

ADDITION TO THE BOSTON CITY HOSPITAL

Construction of the new Pathology Building of the Boston City Hospital was begun on August 15. It is located on the corner of Massachusetts Avenue and Albany Street, and will be four stories high above the basement. As designed by James H. Ritchie and Associates, Architects and Engineers, the building is of modified Georgian style of architecture in keeping with the other recent buildings of the hospital group. The facing will be of brick with stone trimmings.

The first floor will include the ambulance station housing 20 automobiles. There will be an amphitheatre for general meetings with the lower level on the first floor and the upper level extending through the second floor. On the second floor will be the offices of the Medical Examiner and the laboratories for Bacteriology and Serology.

The Pathology laboratories cover the entire third floor. There will be two spacious autopsy rooms on the fourth floor, one of which will be for the use of the Medical Examiner. The stock and experimental animals will be kept on the fourth floor.

Special features of the building include acoustical correctives and flooring materials to reduce noise to the minimum. In the amphitheatre the window light will be controlled from a central point by means of electrically operated light-proof curtain equipment.

The estimated cost of the building is $450,000, and according to present plans it will be completed by April 1, 1933.

SECOND TALK ON TUBERCULOSIS

The second lecture of the series on tuberculosis arranged by the Department of Health and the Bellevue-Yorkville Health Demonstration for physicians was given in the auditorium at 325 East 38th Street on September 21, at 5 P.M. George G. Ornstein, M.D., physician in charge of the tuberculosis division of the Metropolitan Hospital, and director of the department of medicine, Sea View Hospital, spoke on "Sanatorium Care in the Treatment of Tuberculosis." Dr. Ornstein discussed the rôle of the Otisville and Sea View Sanatoria in the city's tuberculosis program. — *Weekly Bulletin City of New York, Department of Health.*

CERTIFICATION OF SPECIALISTS

History repeats itself. A state medical society assumes the responsibility for the certification of specialized practice.

The Medical Society of New Jersey[1] through its house of delegates has recently adopted a plan to accredit its members for special practice. A state committee on credentials has been selected that will determine the qualifications of each applicant. There will be no county or state machinery set up for examination of a man's scientific attainments. Consideration will be given to certification of national examining boards, fellowship in certain national societies, special appointments on hospital staffs, and evidence of continuous active practice during the last ten years, with proof of special ability in their chosen fields of practice. A suitable certificate of qualification carrying the seal of the state medical society and subscribed to by the committee on credentials will be presented to the approved applicants at the annual meeting of the society. A fee of $25 will be charged to defray the cost of certification. The state society reserves the right to revoke any certificate issued, on proof being shown that fraud or misrepresentation was used in obtaining certification.

It is interesting to recall some historical facts[2] regarding the relation of medical societies to licensure. In 1765 the first medical school was founded in Philadelphia, and a year later the first state medical society was organized in New Jersey. In 1772 legislation was secured requiring examination and licensure by two judges of the Supreme Court, with such assistance as they might call of any persons at that date in practice in the colony of New Jersey.

In 1781 the Massachusetts Medical Society was incorporated, and its charter empowered it to make its own rules and regulations "to examine all candidates for the practice of physic and surgery . . . respecting their skill in their profession and . . . if found skilled in their profession and fitted for the

1 J. A. M. A. 99: 397 (July 30) 1932.

2 Waite, F. C.: The Nature of the Responsibility of Medical Licensing Boards, Federation Bulletin 12: 306 (Dec.) 1926.

practice of it they shall receive the approbation of the society in letters testimonial of such examination". The first license was granted in 1782, the same year that the Harvard Medical School was founded.

The New Hampshire Medical Society was chartered in 1791 and was empowered to examine candidates and issue testimonials of proficiency to practice. A new feature was that the society was obligated "to describe and point out such medical instruction or education as they shall judge requisite for the 'Practice of Physic and Surgery' to be completed previous to appearance before them for examination".

The Medical Society of the State of Connecticut received its charter in 1792, which conferred on the society full power to make its own by-laws and regulations, to appoint examining committees in each county to examine candidates, "to license such as shall be found qualified to practice medicine and surgery," and to "confer honorary degrees upon such as they may from time to time find of distinguished merit". This procedure of licensure delegated to the four medical societies mentioned extended to other states, to smaller political subdivisions known as districts, and in some states even to counties.

A hundred years ago the majority of practicing physicians held medical society licenses, frequently called a diploma, and only a minority were medical college graduates. With the formation of sectarian medical societies, the licensing function became more complicated, and about 1835 the granting of licenses was gradually taken away from the medical societies and placed under a state board of medical examiners appointed by the governor. In a number of states the governor is now restricted or influenced in his selection by a list of nominations submitted by the medical societies.

Specialism is an essential part of modern medical practice. In fact, to it must be credited no small part of the advances which have occurred in medical knowledge and practice in recent years. It is recognized, however, that many specialists are self-named; many are not fully trained even in this limited field and still less equipped in the broad fundamentals of medicine. Some are frankly commercial. At present in this country, one-third of recent graduates limit their work to a specialty and most of them have not had a broad clinical experience. Specialism as such is likely to continue to grow, but will gradually become more intimately articulated with the medical needs of the community and more closely coöperative with general medicine and surgery.

It is also becoming more evident that the medical profession has a responsibility of guaranteeing to the public that those who claim to be specialists are in fact expert in their fields, and that recognition be granted only on evidence of the successful completion of a training which, in the opinion of competent medical advice, is adequate to prepare the physician for practice in the limited field.

While it is most important that state medical societies coöperate with the state medical boards in the enforcement of all medical regulatory measures and the disciplining of licensed practitioners, yet to assume the function of certification and qualification of specialists must inevitably lead to confusion in state licensure and registration. Likewise it invites undesirable legal action from disappointed applicants, and certainly dispels all hope of developing a uniform method of regulating specialists applicable to the entire country.

A definite method of qualification was presented at the joint session of the Federation and the Council on Medical Education and Hospitals of the American Medical Association at the educational congress in February, which expressed the best thought of leading specialists, medical educators and state board examiners. It was the general opinion that the regulation of specialists should be of national scope and of uniform application in all states. The function of state boards of licensure and registration should be confined to keeping a registry of specialists qualified in accordance with the regulations to be generally adopted. National boards of examiners in the different fields of special practice, properly constituted, can generally be accepted to best determine the qualifications necessary in each particular specialty.

The great need of the moment is to secure joint leadership in a national program that will embody the educational ideals and methods of the university and the highest types of specialized practice.—*Federation Bulletin.* Federation of State Medical Boards of the United States.

PHARMACOPOEIAL ADMISSIONS AND DELETIONS

The Sub-Committee on Scope, of the Committee of Revision of the U.S.P.XI has announced its first decisions on what is commonly spoken of as "Admissions and Deletions". The members of the Sub-Committee have diligently pursued their part in the work of revision and their discussions and conclusions cover almost 400 pages of Sub-Committee Bulletins. In addition to this, the Sub-Committee has held three personal conferences when titles were discussed and many decisions reached.

The members of the Sub-Committee are: Dr. Reid Hunt, Chairman, with Messrs. Bastedo, Beardsley, Bethea, Brown, Christian, Dooley, DuMez, Edmunds, Fantus, Hirschfelder, Lascoff, LaWall, McCoy, Nelson, Roth, Seltzer, Simpson, Starr, Tatum, Underhill*, H. C. Wood, Jr., and C. B. Wood.

The following lists are announced for the information of members of the medical and pharmaceutical professions and the General Chairman invites a free discussion of these first decisions. Comments re-

*Deceased, June 28, 1932.

ceived by him will be issued in the official circulars under the author's name and referred to the Sub-Committee for their consideration. General statements are of little help to the Committee. Criticisms or comments should deal with specific items and offer, if possible, facts to support the opinion expressed.

DELETIONS PROPOSED

The following U.S.P.X. titles, 108 in number, have not been admitted to the U.S.P.XI:

Aconitina, Antitoxinum Tetanicum Crudum, Benzaldehydum, Buchu, Fluidextractum Buchu, Calcii Glycerophosphas, Calumba, Tinctura Calumbae, Cambogia, Cimicifuga, Fluidextractum Cimicifugae, Cinchonidinae Sulphas, Colchici Cormus, Extractum Colchici (Cormi), Fluidextractum Colchici (Seminis), Colocynthis, Extractum Colocynthidis, Extractum Colocynthidis Compositum, Cotarninae Chloridum, Cubeba, Elaterinum, Emplastrum Capsici, Emplastrum Plumbi Oleatis, Eucalyptus, Fluidextractum Eucalypti, Ferri Carbonas Saccharatus, Ferri Chloridum, Ferri Phosphas Solubilis, Ferri Sulphas Exsiccatus, Fluidextractum Belladonnae Foliorum, Fluidextractum Cinchonae, Fluidextractum Hyoscyami, Fluidextractum Rhei, Fluidextractum Scillae, Gambir, Tinctura Gambir Composita, Glyceritum Phenolis, Granatum, Fluidextractum Granati, Guaiacolis Carbonas, Hydrargyri Iodidum Rubrum, Hydrastis, Fluidextractum Hydrastis, Hyoscyaminae Hydrobromidum, Infusum Digitalis, Ipomoeae, Resina Ipomoeae, Jalap, Resina Jalapae, Krameria, Tinctura Krameriae, Linimentum Calcis, Liquor Arsenii et Hydrargyri Iodidi, Liquor Ferri et Ammonii Acetatis, Liquor Plumbi Subacetatis, Liquor Potassii Arsenitis, Liquor Potassii Citratis, Liquor Potassii Hydroxidi, Liquor Sodae Chlorinatae, Liquor Sodii Hydroxidi, Liquor Zinci Chloridi, Lobelia, Tinctura Lobeliae, Manna, Mistura Glycyrrhizae Composita, Morphinae Hydrochloridum, Oleoresina Capsici, Oleum Cajuputi, Oleum Cari, Oleum Tiglii, Paraformaldehydum, Pepo, Phosphorus, Pilocarpinae Hydrochloridum, Pilulae Asafoetidae, Pilulae Hydrargyri Chloridi Mitis Compositae, Pilulae Phosphori, Plumbi Monoxidum, Pulvis Jalapae Compositus, Pulvis Rhei Compositus, Quassia, Quininae Hydrobromidum, Quininae Hydrochloridum, Quininae Tannas, Rhus Glabra, Fluidextractum Rhois Glabra, Salicinum, Senega, Fluidextractum Senegae, Syrupus Senegae, Syrupus Scillae Compositus, Strontii Salicylas, Strophanthus, Tinctura Strophanthi, Sulphonmethanum, Syrupus Rhei, Tinctura Asafoetidae, Tinctura Cardamomi, Tinctura Cinchonae, Tinctura Rhei, Tinctura Valerianae Ammoniata, Trochisci Acidi Tannici, Trochisci Ammonii Chloridi, Ulmus, Unguentum Iodoformi, Unguentum Plumbi Oleatis, Uva Ursi, Fluidextractum Uvae Ursi.

NEW ADMISSIONS

The following new admissions have also been announced:

Acriflavine, Antimeningococcus Serum, Antipneumococcus Serum—Type 1, Antiseptic Iodine Solution (exact formula to be determined), Carbon Dioxide, Chiniofon (Sodium iodoxyquinoline sulphonate), Digitalis Solution for Injection, Diphtheria Toxin for Schick Test, Diphtheria Toxoid, Emulsion of Mineral Oil, Ephedrine, Ephedrine Sulphate, Ethylene for anesthesia, Fluorescein, Histamine Acid Phosphate, Solution of Histamine Acid Phosphate, Iron Arsenate (standardize for Ampul Manufacture), Iron Arsenite (standardize for Ampul Manufacture), Iron Citrate (Green) (standardize for Ampul Manufacture), Liver Extract, Phenobarbital Soluble, Rabies vaccine, Tetraiodo-phenolphthalein Sodium, Tuberculin—Old, Typhoid Vaccine.

A few additional titles have been tentatively admitted, subject to the adjustment of possible patent or trade mark complications. In this list are Insulin and Viosterol and other similarly involved substances. It is hoped that arrangements can be made for the admission of most of these.

Comments should be sent to the General Chairman.

E. Fullerton Cook,
43rd Street and Woodland Avenue,
Philadelphia, Pa.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS

For August, 1932

Anterior poliomyelitis has continued to be exceptionally low, the number of cases being the lowest ever reported for the month of August.

Typhoid fever showed a decrease over previous years, and the number of cerebrospinal meningitis cases reported was below the usual prevalence expected for this month.

Whooping cough, chicken pox, lobar pneumonia, German measles, mumps and influenza were reported about the number of times expected during August.

Diphtheria continued to show a downward trend. The number of cases reported was the lowest ever recorded for any month in the history of the Department.

Measles and scarlet fever showed a decrease over July, but both are about normal for this time of year.

RARE DISEASES

Anterior poliomyelitis was reported from Adams, 1; Boston, 4; Fall River, 1; Greenfield, 1; Sheffield, 1; Upton, 1; Out of State, 1; Total, 10.

Encephalitis lethargica was reported from Pittsfield, 1; Salem, 1; Worcester, 1; Total, 3.

Epidemic cerebrospinal meningitis was reported from Boston, 2.

Pellagra was reported from Worcester, 1.

Septic sore throat was reported from Boston, 2; Cambridge 1; Haverhill, 1; Lowell, 1; Quincy, 1; Winthrop, 1; Total 7.

Tetanus was reported from Out of State, 1.

Trachoma was reported from Boston, 1; Medford, 1; Weymouth, 1; Worcester, 1; Total, 4.

Trichinosis was reported from Belmont, 1; Boston, 3; Lynn, 1; Somerville, 1; Watertown, 2; Total, 8.

Undulant fever was reported from Amherst, 1; Greenfield, 1; Total, 2.

MONTHLY REPORT OF CERTAIN COMMUNICABLE DISEASES

	Cases in Entire Pop.				Case Rates per 100,000 Pop.		
	August 1932	August 1931	Proso. Index*	Epi. Index†	August 1932	August 1931	Expected Rates‡
All Causes	3718	4498	—	—	86.5	105.2	—
Ant. Polio.	10	433	110	.1	.2	10.1	2.6
Diphtheria	121	131	191	.6	2.8	3.1	4.4
Ep. C. S. Men.	2	7	5	.4	.04	.2	.1
Measles	254	179	329	.8	5.9	4.2	7.7
Pneumonia Lobar	75	73	71	1.0	1.7	1.7	1.7
Scarlet Fever	317	290	233	1.4	7.4	6.8	5.4
Tuberculosis Pulmonary	299	302	348	.9	7.0	7.1	8.1
Typhoid Fever	29	33	33	.9	.7	.8	.8
Whooping Cough	254	557	694	.7	10.6	13.0	16.2
Chicken Pox	123	112	—	—	2.9	2.6	—
German Measles	26	39	—	—	.6	.9	—
Gonorrhea	653	679	—	—	15.2	15.9	—
Influenza	5	8	—	—	.1	.2	—
Mumps	176	185	—	—	4.1	4.3	—
Syphilis	333	398	—	—	7.7	9.3	—
Tuberculosis. Other Forms	34	37	—	—	.8	.9	—

*This index is an attempt to estimate the number of cases based on the trend during the past years which can be expected to occur, and is for the purpose of comparison with the number of cases which actually did occur.

†This ratio expresses how prevalent the disease is compared with the index mentioned above; 1.0 indicates that the actual number of cases equals the expected number. A larger number means a greater prevalence, and a smaller number a lesser prevalence than expected. Thus 2.0 would indicate twice the expected number, and .5 half the expected number of cases. The method used to determine the indices is described in the August 18, 1927 issue of the *Boston Medical and Surgical Journal*.

‡Calculated from the Prosodemic Index.

RECENT DEATHS

SYLVESTER — STEPHEN A. SYLVESTER, M.D., formerly a practitioner in Newton, Massachusetts, died September 18, 1932, in Lakeland, Florida, where he had lived since his retirement from practice in 1911. He was born in New Ipswich, New Hampshire, in 1848. His medical education was acquired at the Boston University School of Medicine from which he graduated in 1875. He was twice married, first to Mary Louise Haskell of Fitchburg, Massachusetts, who died in 1918. Subsequently he married Letitia Vertress of Lakeland, Flordia, who survives him. He also left two sons Dr. Philip H. Sylvester of 25 Bay State Road, Boston, and Carl A. Sylvester of Rio de Janeiro, Brazil, and a half brother William H. Sylvester of Lawrence, Massachusetts.

SWAN—WILL HOWARD SWAN, M.D., died in Colorado Springs, Colorado, September 5, 1932, aged 67. Dr. Swan was a former Fellow of the Massachusetts Medical Society retiring in 1901 after moving to Colorado Springs in 1900. He was a graduate of the Harvard University Medical School class of 1891. He was born in Winchester, New Hampshire, and after the completion of his medical course he practiced in Beverly, Massachusetts, for six years later moving to Colorado Springs where he practiced for thirty years until he retired two years ago.

Dr. Swan is survived by his widow who was Miss Anna Creelman of Beverly and two daughters Miss Catherine C. Swan of New York City and Miss Dorothy Swan of Colorado Springs.

OBITUARY

DR. WILLIAM E. FAULKNER

In the death of Dr. William E. Faulkner, which occurred at his home in Keene on September 16, his many personal friends and professional associates, especially in Boston, where most of his professional work was done, have met with a loss that they will feel severely. His place in their hearts will be indeed hard to fill. He united to a charming personality a keen sense of humor, a healthy love of outdoor sports, and a genius for friendship. He never entered into the rough and tumble of the struggle for practice. He did not have to. His professional work, however, at the Boston City Hospital for twenty years was characterized by faithfulness, good judgment and skill. He gave the patients at that hospital for all those years the very best that was in him. He was prodigal of his time and strength. He was ready at any time to take on the work of fellow members of the staff who were in need of relief in their hospital work.

A son of Charles S. and Sarah Eliza (Eames) Faulkner, he was born in Keene, November 7, 1863. He attended the Keene and Boston schools where he prepared for Harvard, graduating in 1887 with the B.A. degree. He received his M.D. degree at Harvard in 1890. He spent a year and a half as

surgical house officer in the Boston City Hospital and then continued his studies in Vienna and Berlin, and on his return to the United States he was a visiting surgeon at the Boston City Hospital, a position he maintained for twenty years. He retired from active practice in 1922.

In 1917 he built a country home in Keene, on a hill having a view of Mt. Monadnock, and since his retirement had lived there in the fullest enjoyment of life in that lovely country.

When the World War broke out Dr. Faulkner had a great desire to enter the conflict and joined the British Army, going to France with the first detachment of Base Hospital No. 22, the Harvard Unit, in June, 1915, and remaining in France until 1917. When the United States entered the war he joined our army as a Major and was in charge of the surgical division of base hospitals in Ann Arbor and Texas. His service in war as in peace was ungrudging, faithful and cheerfully given.

On July 7, 1906, in Geneva, Switzerland, he married Rosalie Edwards of Philadelphia, Pa., a daughter of John and Lydia Bowman (Baker) Edwards, who survives; also four sons, William E. Faulkner, Jr., Frederick Faulkner, Edmund Faulkner, a Harvard student, and Robert E. Faulkner; two brothers, Dr. Herbert K. Faulkner, Robert E. Faulkner, and a sister, Miss Mary Faulkner; all of Keene. He also leaves several nephews and nieces.

He was a member of the American College of Surgeons, American Surgical Association, New England Surgical Society, and American Medical Association. He was consulting surgeon on the staff of the Elliot Community Hospital in Keene for many years. He was a director of the Faulkner and Colony Manufacturing Company in Keene.

Socially he was most delightful and his company was often sought by his professional friends and classmates, who after his retirement often enjoyed his hospitality at his home in Keene. One of his deepest friendships was with the late Dr. Charles Allen Porter. He was a real athlete, playing tennis well almost to the end of his life, and was an enthusiastic mountain climber. After he left Vienna and before entering practice he traveled extensively in the pursuit of big game in India.

As will be seen from the above inadequate account, his activities outside his professional career were extensive, and of a character most wholesome. His family life was ideal; his professional work most conscientious, thorough and devoted. His friends and family may well be proud of his character and his record.

F. B. L.

NOTICES

APPOINTMENT

Sanitary Engineer L. M. Fisher. Directed to proceed from New York City to points in Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey and Pennsylvania, and return, during September, 1932, in connection with certification of water supplies used on interstate carriers.

ANNOUNCEMENT

Dr. Rockwood H. Thayer will not be associated with the Holden Clinic after October first. He is to open an office at 27 Elm Street, Worcester.

REMOVAL

Robert H. Nichols, M.D., announces the removal of his office to 45 Bay State Road, Boston.

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P.M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The first clinic will be given on October 6, 1932.

Also physicians and medical students are invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays.

REPORTS AND NOTICES OF MEETINGS

THE FOUR COUNTY MEDICAL SOCIETY

The annual meeting of the "Four County Medical Society" of Western Massachusetts was held September 14, 1932, at the New Springfield Hospital. The following program was a departure from the social gatherings formerly held:

9:00 to 11:00 A.M.

Operating Rooms Surgical Clinics

10:30 A.M.

DEMONSTRATIONS

Presentation of Hospital Cases.

10:30 Medical: Dr. M. J. Stoddard, Dr. R. L. Barrett.

10:45 Pediatrics: Dr. W. B. Adams.

11:00 Orthopedic: Dr. H. L. Wheat.

11:15 Surgical: Dr. F. B. Sweet, Dr. F. D. Jones.

11:30 A.M.

PAPERS

11:30 Streptococcus Pneumonia and Some of Its Complications, Dr. A. E. Johnson, Jr., Greenfield.

11:45 Some Observations on Trans-Urethral Resection of Prostate (with lantern slides), Dr. Nathan Finklestein, Pittsfield.

12:00 Surgical Tonsillectomy vs. Electro-coagulation, Dr. E. G. Boss, Springfield.

12:15 A Case of Traumatic Rupture of Gall Bladder, Dr. P. E. Gear, Holyoke.

12:30 P.M.—Inspection of Building.

1:30 P.M.—Dinner. Main Dining Room.

Welcome—Dr. Eugene Walker, Superintendent, Springfield Hospital.

Problems of the State Society, Dr. H. G. Stetson, Greenfield, President, State Society.

The General Practitioner's Need for More Careful Analysis of Cases of Chronic Rheumatism, Dr. R. B. Osgood, Boston.

J. A. WHITNEY, M.D., *Secretary.*

WATERTOWN PHYSICIANS' SOCIETY

The regular monthly meeting of the Watertown Physicians' Society will be held Monday, October 3, 1932, 8:30 P.M., at 128 Mt. Auburn Street, Watertown.

Dr. Lester R. Whitaker will speak on "Some Newer Aspects of Diagnosis in Gall Bladder Disease."

Interested physicians are cordially invited to attend.

EUGENE F. GORMAN, *Secretary.*

HAMPDEN DISTRICT MEDICAL SOCIETY

The regular Fall Meeting of the Society will be held at the Monson State Hospital, Monson, Mass., on Tuesday, October 18, 1932. The members of the Society will be guests of Dr. M. B. Hodskins, Superintendent of the Hospital.

Complete program will be published later.

H. L. SMITH, *Secretary.*

WILLIAM HARVEY SOCIETY

The opening meeting of the William Harvey Society, of the Tufts College Medical School, will be held at 8 o'clock P.M. on October 7 at the Beth Israel Hospital. The speaker will be Dr. Francis Carter Wood, Director of the Pathological Department of St. Luke's Hospital of New York City, Director of the Institute of Cancer Research at Columbia University, Editor of the *American Journal of Cancer*, and Reviser of the Textbook of Pathology by Delafield and Prudden. His subject will be "The Research Side of Cancer," illustrated by motion picture film.

Dr. Harold E. MacMahon, Professor of Pathology and Bacteriology at the Tufts College Medical School, will be the Chairman of the meeting.

Medical students and members of the medical profession are cordially invited.

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

September 30, October 1—Scientific Program for Annual Meeting of the New England Surgical Society. See page 333, issue of August 18.

October 3—Watertown Physicians' Society. See notice above.

October 6—Clinic in Medicine at Peter Bent Brigham Hospital. See notice on page 593.

October 7—William Harvey Society. See notice above.

October 12, 13 and 14—American Academy of Physical Therapy. See notice on page 384, issue of August 25.

October 17-21—Clinical Congress of the American College of Surgeons. See page 1330, issue of June 23.

October 17-28—The 1932 Graduate Fortnight of the New York Academy of Medicine. See page 1375, issue of June 30.

October 19—The New England Dermatological Society. See notice on page 557, issue of September 22.

October 19—The New England Physical Therapy Society. First meeting of the season. Detailed notice will appear in a later issue of the Journal.

October 22 and December 7—American Board of Obstetrics and Gynecology. Announcement of Coming Examinations. See notice on page 557, issue of September 22.

October 22-24—Health Education Institute. See notice on page 47, issue of July 7.

October 24-27—American Public Health Association. See notice on page 1321, issue of December 31, 1931.

October 24-28—Inter-state Postgraduate Medical Association of North America. See page 39, issue of January 7.

DISTRICT MEDICAL SOCIETIES

HAMPDEN DISTRICT MEDICAL SOCIETY

October 18—See notice elsewhere on this page.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

November 9 at Winchester.
January 11 at Wakefield.
March 15 at Melrose.
May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

Schedule of meetings for 1932-33:

Wednesday, October 12—Grafton State Hospital. Dinner and program consisting of short papers on "Contagion," to be followed by a talking picture to be selected later.

Wednesday, November 9—Dinner and scientific meeting at the Worcester Hahnemann Hospital. Program to be announced later.

Wednesday, December 14—Meeting at the Worcester Chamber of Commerce Auditorium. Paper by Dr. Joseph Meigs of Boston, entitled "Uterine Bleeding."

Wednesday, January 11—Dinner and meeting at the Worcester City Hospital. Paper by Dr. Morris Fishbein, Editor of the Journal of the American Medical Association.

Wednesday, February 8—Dinner and scientific meeting at the Worcester State Hospital. Program to be announced later.

Wednesday, March 8—Dinner and scientific program at the Memorial Hospital, Worcester. Program to be announced later.

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, Secretary.

BOOK REVIEW

The Aetiology of Tuberculosis. By DR. ROBERT KOCH. Published by The National Tuberculosis Association. Price 50c.

This booklet is an attractively prepared reprint of Dr. Robert Koch's epoch-making paper, in which he gave the results of his investigations which led to the discovery of the tubercle bacillus. This was originally read before the Physiological Society in Berlin, March 24, 1882, and published in the *Berliner Klinische Wochenschrift* of that date.

The National Tuberculosis Association, believing that at this time, the 50th anniversary of Dr. Koch's discovery, there was a widespread desire for an easily accessible textbook for English readers, presented this textbook as a fitting memorial of the semi-centennial anniversary of the great discovery.

The introduction by Dr. Allen K. Krause is a masterly piece of work, reviewing the events and factors which led up to this discovery. This adds enormously to the value of the translation of the original article which follows. No more fitting tribute to the memory of a great man can be imagined.